This is a digital copy of a book that was preserved for generations on library shelves before it was carefully scanned by Google as part of a project to make the world's books discoverable online.

It has survived long enough for the copyright to expire and the book to enter the public domain. A public domain book is one that was never subject to copyright or whose legal copyright term has expired. Whether a book is in the public domain may vary country to country. Public domain books are our gateways to the past, representing a wealth of history, culture and knowledge that's often difficult to discover.

Marks, notations and other marginalia present in the original volume will appear in this file - a reminder of this book's long journey from the publisher to a library and finally to you.

Usage guidelines

Google is proud to partner with libraries to digitize public domain materials and make them widely accessible. Public domain books belong to the public and we are merely their custodians. Nevertheless, this work is expensive, so in order to keep providing this resource, we have taken steps to prevent abuse by commercial parties, including placing technical restrictions on automated querying.

We also ask that you:

+ *Make non-commercial use of the files* We designed Google Book Search for use by individuals, and we request that you use these files for personal, non-commercial purposes.

+ *Refrain from automated querying* Do not send automated queries of any sort to Google's system: If you are conducting research on machine translation, optical character recognition or other areas where access to a large amount of text is helpful, please contact us. We encourage the use of public domain materials for these purposes and may be able to help.

+ *Maintain attribution* The Google "watermark" you see on each file is essential for informing people about this project and helping them find additional materials through Google Book Search. Please do not remove it.

+ *Keep it legal* Whatever your use, remember that you are responsible for ensuring that what you are doing is legal. Do not assume that just because we believe a book is in the public domain for users in the United States, that the work is also in the public domain for users in other countries. Whether a book is still in copyright varies from country to country, and we can't offer guidance on whether any specific use of any specific book is allowed. Please do not assume that a book's appearance in Google Book Search means it can be used in any manner anywhere in the world. Copyright infringement liability can be quite severe.

About Google Book Search

Google's mission is to organize the world's information and to make it universally accessible and useful. Google Book Search helps readers discover the world's books while helping authors and publishers reach new audiences. You can search through the full text of this book on the web at `http://books.google.com/`

LANE MEDICAL LIBRARY STANFORD
L74 .J33 1904 STOR
The clinical study of blood-pressure : a
24503417798

LANE
MEDICAL LIBRARY
SEAL OF THE LELAND STANFORD JUNIOR UNIVERSITY
1885
GIFT
Dr. Wm.Fitch Cheney

LANE LIBRARY. STANFORD UNIVERSITY

THE CLINICAL STUDY OF BLOOD-PRESSURE

THE
CLINICAL STUDY OF BLOOD-PRESSURE

A GUIDE TO THE USE OF THE

SPHYGMOMANOMETER

IN MEDICAL, SURGICAL, AND OBSTETRICAL PRACTICE, WITH A SUMMARY
OF THE EXPERIMENTAL AND CLINICAL FACTS RELATING TO
THE BLOOD-PRESSURE IN HEALTH AND IN DISEASE

BY

THEODORE C. JANEWAY, M. D.
LECTURER ON MEDICAL DIAGNOSIS, UNIVERSITY AND BELLEVUE HOSPITAL
MEDICAL COLLEGE, AND VISITING PHYSICIAN TO
CITY HOSPITAL, NEW YORK CITY

SEVENTY-FIVE ILLUSTRATIONS IN THE TEXT
MANY IN COLORS

LANE LIBRARY. STANFORD UNIVERSITY

NEW YORK AND LONDON
D. APPLETON AND COMPANY
1907

Copyright, 1904, by
D. Appleton and Company

Printed at the Appleton Press
New York, U. S. A.

J 35
1904

TO

MY FATHER

MY BEST AND WISEST TEACHER
FROM CHILDHOOD UNTIL THIS DAY
I DEDICATE
WHATEVER IS WORTHY
IN THIS BOOK

47663

PREFACE

THE modern physician's armamentarium is so complex that additions to it are far from an unmixed boon. Nevertheless, the sphygmomanometer has been welcomed in many quarters as an instrument of real value. Two questions, however, must be fairly and squarely answered, before its general use can be advocated; First, Does it yield accurate information, which can be had in no easier way? Second, Is this information worth the time expended in obtaining it?

The answer to the first question is simple. Five minutes' trial will convince the most sceptical that his previous judgments, based on his supposedly trained sense of touch, were often fallacious. High tension was certainly recognized before the introduction of the sphygmomanometer, but so was fever before the days of clinical thermometers. No physician would endorse the sentiments of the old colored mammy, who said, "What you want wif dese new-fangle fermometers, honey? I puts de babies in de baf, and if de water's too cole it turns 'em blue, if it's too hot it turns 'em red." In medicine, accuracy of observation is the first step toward a correct diagnosis, without which nothing but bald empiricism is possible.

The second query cannot have so prompt a reply. The value of a knowledge of the actual blood-pressure in a particular case must depend largely on the observer's acquaintance with the physiological and pathological causes for variation. It is the aim of the author to make this knowledge, which is now scattered through text-books of physiology, pathology, and practice, and a considerable journal literature of twenty odd years, readily available to every practitioner. With this information, both diagnosis, prognosis, and therapeutics cannot but gain in efficiency through blood-pressure determinations, at the bed-side or in the office.

His personal conviction of the truth of this statement, after the use of various sphygmomanometers and some critical study of their value, must be his excuse for the presentation of this book. In its preparation he has incurred many obligations, which he can only acknowledge here with hearty thanks; to his father, Dr. E. G. Janeway, for most of the opportunities for clinical study; to his friends, Prof. Graham Lusk, Drs. E. M. Evans, Stuart Hart, and Horst Oertel, for personal assistance; to Dr. H. W. Cook, for his courtesy in allowing the reproduction of his valuable charts, and to Dr. J. Erlanger, for the mechanical drawings of his sphygmomanometer. In the literature, on the physiological side, the invaluable text-book of Tigerstedt; on the technical side, the work of v. Recklinghausen, of Gumprecht, and of Vaschide and Lahy; and in the clinical portion, the articles of Cook and Briggs, Hensen, and Cushing, and Crile's studies, have been of the greatest service. He would also thank Drs. Province, W. C. Garvin Higgins, A. H. Garvin, Chapin, Boyd, and Garside, of the House Staff of City Hospital, for their aid and coöperation in obtaining illustrative charts, and Mr. Ed. Ruschli and Mr. Max Jütte, of the second year class at the University and Bellevue Hospital Medical College, the former for manometric tracings, the latter for aid in preparing part of the bibliography. The excellent drawings of apparatus are by Mr. K. K. Bosse.

T. C. J.

36 West Fortieth Street.

TABLE OF CONTENTS

PART I.—PHYSIOLOGICAL

CHAPTER PAGE

I.—THE DIRECT MEASUREMENT OF BLOOD-PRESSURE 1
1. Introduction—The mercurial manometer 3
2. The normal manometric trace. 4
3. The errors of the mercurial manometer—Compensated manometer 5
4. Elastic manometers—Maximum and minimum manometers. . 6

II.—BLOOD-PRESSURE IN THE NORMAL ANIMAL 7
1. General features of the arterial circulation 9
2. The factors which determine blood-pressure 11
3. The mean aortic blood-pressure in different animals . . . 28
4. The blood-pressure in different arteries 30
5. The periodic variations of aortic blood-pressure 32
6. Spontaneous non-rhythmical variations of aortic blood-pressure . 37
7. The influence of asphyxia on blood-pressure 37
8. The influence of atmospheric pressure on blood-pressure . . 38
9. The influence of muscular work on blood-pressure . . . 39

PART II.—TECHNICAL

III.—THE INDIRECT MEASUREMENT OF BLOOD-PRESSURE 41
1. Introduction 43
2. The development of sphygmomanometers applying pressure to the artery through a fluid medium 46
3. The method of circular compression by air 54
4. Experimental verification of the method of circular compression. 55
5. Validity of the several criteria 62

IV.—THE MODERN SPHYGMOMANOMETERS 75
1. Introduction 77
2. Instruments measuring systolic pressure only 77
3. Instruments measuring diastolic pressure 84
4. Instruments measuring systolic pressure and affording an approximate estimate of diastolic pressure 87
5. Instruments measuring accurately both systolic pressure and diastolic pressure by graphic record 93
6. Comparison of results obtained with the various sphygmomanometers 100
7. Blood-pressure records 101
8. Practical suggestions 102

PART III.—CLINICAL

CHAPTER PAGE
V.—BLOOD-PRESSURE IN THE NORMAL MAN 105
1. General considerations 107
2. Blood-pressure in different individuals 108
3. Blood-pressure variations in the same individual . . . 112
Literature 129

VI.—THE BLOOD-PRESSURE IN DISEASE 133
1. Pathological limits of blood-pressure 135
2. High blood-pressure (hypertension) 137
3. Low blood-pressure (hypotension) 152
4. The relation of blood-pressure to pulse-rate and temperature . 160
5. Blood-pressure as a guide to the functional ability of the heart and the adequacy of the circulation 161
Literature 163

VII.—BLOOD-PRESSURE IN INTERNAL DISEASES 167
1. Diseases of the heart, arteries, and kidneys 169
Literature 214
2. Acute infectious diseases 216
3. Chronic infectious diseases 231
4. Chronic diseases associated with impaired nutrition . . . 235
5. Miscellaneous 238
Literature 242

VIII.—BLOOD-PRESSURE IN NERVOUS AND MENTAL DISEASES . . . 245
1. Diseases of the spinal cord 247
2. Diseases of the brain 248
3. The psychoses 254
4. Functional conditions 256
Literature 258

IX.—BLOOD-PRESSURE IN SURGICAL CONDITIONS 259
1. Uses of the sphygmomanometer in surgical practice . . . 261
2. Blood-pressure during surgical operations 261
3. Blood-pressure in surgical accidents and diseases . . . 272
Literature 278

X.—BLOOD-PRESSURE IN OBSTETRICAL CONDITIONS 281
1. Value of the sphygmomanometer in obstetrical practice . . 283
2. Blood-pressure during pregnancy 284
3. Blood-pressure during labor and the puerperium . . . 285
4. Puerperal eclampsia 287
Literature 289

LIST OF ILLUSTRATIONS

FIGURE PAGE

1. *Tracing.*—The normal manometric trace 5
2. *Diagram.*—The flow of liquid through tubes 10
3. *Tracing.*—Fall in blood-pressure produced by stimulation of the peripheral cut end of the vagus 12
4. *Tracing.*—Fall in blood-pressure produced by stimulation of the central cut end of the vagus (depressor effect) . . . 20
5. *Tracing.*—Rise in blood-pressure produced by stimulation of the anterior crural nerve (pressor effect) 21
6. *Tracing.*—Fall in blood-pressure produced by hæmorrhage . . . 27
7. *Cut.*—v. Basch's sphygmomanometer. First model 46
8. *Cut.*—v. Basch's sphygmomanometer. Recent model 47
9. *Cut.*—Potain's sphygmomanometer 48
10. *Cut.*—Marey's second sphygmomanometer 51
11. *Cut.*—Mosso's sphygmomanometer 52
12. *Diagram.*—Cross-section of arm, Riva-Rocci armlet 54
13. *Diagram.*—Cross-section of arm, Hill and Barnard armlet 54
14. *Diagram.*—Cross-section of finger with Gärtner ring 55
15. *Diagram.*—Distortion of a tube by blocks laid on it 57
16. *Diagram.*—Manner of closure, normal and sclerotic artery . . . 60
17. *Tracing.*—Tracings from the forearm, illustrating the return of the pulse under compression 63
18. *Tracing.*—The pulsations transmitted through an elastic membrane separating two vessels 70
19. *Diagram.*—Apparatus of Walden for verifying the criterion of maximum pulsation of the arterial wall 71
20. *Tracing.*—Normal pulse, Erlanger's sphygmomanometer . . . 72
21. *Cut.*—Riva-Rocci's sphygmomanometer 79
22. *Cut.*—Cook's sphygmomanometer 81
23. *Cut.*—Gärtner's tonometer 83
24. *Cut.*—Hill and Barnard's sphygmometer 85
25. *Cut.*—Stanton's sphygmomanometer 88
26. *Cut.*—Author's sphygmomanometer 90
27. *Cut.*—Erlanger's sphygmomanometer 94
28. *Cut.*—Erlanger's sphygmomanometer, stop-cock from above . . 95
29. *Cut.*—Erlanger's sphygmomanometer, section of stop-cock, position "IN" 96

FIGURE PAGE

30. *Cut.*—Erlanger's sphygmomanometer, section of stop-cock, position "OUT" 97
31. *Cut.*—Erlanger's sphygmomanometer, section of stop-cock, position 1 97
32. *Tracing.*—High tension pulse, Erlanger's sphygmomanometer . . 99
33. *Chart.*—Illustrating manner of recording blood-pressure, pulse, and respiration. Case of lobar pneumonia . . *Facing* 102
34. *Tracing.*—Periodic fluctuations of blood-pressure, healthy man . . 113
35. *Chart.*—Diurnal blood-pressure chart of a healthy man . . . 114
36. *Chart.*—Diurnal variations of diastolic pressure, the influence of meals being excluded. 115
37. *Chart.*—Rise in blood-pressure produced by tobacco 118
38. *Chart.*—Rise in blood-pressure produced by mental effort . . . 120
39. *Chart.*—Rise in blood-pressure produced by muscular exertion . . 121
40. *Chart.*—Influence of muscular exertion on blood-pressure in the aged 123
41. *Chart.*—Influence of water-baths of different temperatures on blood-pressure and pulse-rate 125
42. *Chart.*—Marked hypertension in chronic interstitial nephritis . . 170
43. *Chart.*—Chronic nephritis; left hemiplegia; lobar pneumonia; death 174
44. *Chart.*—Chronic uræmia 180
45. *Chart.*—General arterio-sclerosis 186
46. *Tracing.*—Pulsus differens. Case of obstruction of the left axillary artery 190
47. *Chart.*—Aortic insufficiency 196
48. *Chart.*—Terminal hypotension in cardiac disease 199
49. *Chart.*—Œdema of the lungs 206
50. *Cut.*—Dehio's apparatus 209
51. *Chart.*—Fall in blood-pressure produced by nitroglycerin, gr. $\frac{1}{100}$. 211
52. *Chart.*—Fall in blood-pressure produced by nitroglycerin, gr. $\frac{3}{50}$ . . 212
53. *Chart.*—Typhoid fever (showing effect of baths) 218
54. *Chart.*—Typhoid fever—intestinal hæmorrhage 219
55. *Chart.*—Typhoid fever—intestinal perforation 220
56. *Chart.*—Typhoid fever with toxæmic features and hypotension (showing effect of digitalin) 222
57. *Chart.*—Absence of any rise in blood-pressure after the subcutaneous injection of adrenalin 223
58. *Chart.*—Extreme rise in blood-pressure produced by the intravenous injection of adrenalin 224
59. *Chart.*—Fatal lobar pneumonia 228
60. *Chart.*—Marked hypotension in acute tuberculosis 232
61. *Chart.*—Chronic pulmonary tuberculosis with slight hypotension . 233
62. *Chart.*—Intracranial hæmorrhage (apoplexy) with exploration of the brain and evacuation of a large clot 250
63. *Chart.*—Relation between blood-pressure and psychical state in melancholia 254
64. *Chart.*—Average curves of blood-pressure during anæsthesia . . 262
65. *Chart.*—Thoracotomy for post-pneumonic empyema under local cocain anæsthesia 264

FIGURE PAGE
66. *Chart.*—Excision of hydrocele sac after spinal cocainization . . . 265
67. *Chart.*—Forcible stretching of left sciatic nerve for sciatica . . . 266
68. *Chart.*—Operation for osteomyelitis of femur 267
69. *Chart.*—Laparotomy (appendectomy) 268
70. *Chart.*—Cholecystectomy . . . . *Facing* 269
71. *Chart.*—Laparotomy for uterine fibroids 270
72. *Chart.*—Prostatectomy . . . . *Facing* 272
73. *Chart.*—Traumatic shock without hæmorrhage 274
74. *Chart.*—Post-operative depression (twin labor) 286
75. *Chart.*—Puerperal eclampsia 288

ABBREVIATIONS USED

(v. B.) = v. Basch's sphygmomanometer (see page 46).
(Erl.) = Erlanger's sphygmomanometer (see page 93).
(G.) = Gärtner's tonometer (see page 82).
(H. & B.) = Hill and Barnard's sphygmometer (see page 84).
(J.) = Author's sphygmomanometer (see page 89).
(Mosso) = Mosso's sphygmomanometer (see page 51).
(P.) = Potain's sphygmomanometer (see page 48).
(R. R.) = Riva-Rocci's sphygmomanometer, or Cook's modification (see pages 78 and 80).

(5 cm.), (12 cm.), etc., refer to the width of compressing surface in the armlet used.
(1.5 cm.) or (2.5 cm.) refer to the width of compressing surface in the Gärtner ring used.

Sys. = Systolic end pressure.
Diast. = Diastolic lateral pressure.

PART I.—PHYSIOLOGICAL

CHAPTER I

THE DIRECT MEASUREMENT OF BLOOD-PRESSURE

1. Introduction.

- A. The mercurial manometer.
- B. The kymographion.

2. The normal manometric trace.

3. The errors of the mercurial manometer—Compensated manometer.

4. Elastic manometers—Maximum and minimum manometers.

CHAPTER I

THE DIRECT MEASUREMENT OF BLOOD-PRESSURE

1. INTRODUCTION

THAT the blood exerts pressure upon the vessels through which it circulates is, of course, a necessary corollary of the fact that it flows; but more than a hundred years elapsed before Harvey's discovery of the circulation was followed by Hales's[1] demonstration of the blood-pressure, in 1733, and a second century before its accurate study was begun.

A. The Mercurial Manometer.—The exact determinations which mark the beginning of modern physiological experiment took their origin with two men, Poiseuille and Ludwig. The former, in 1828,[2] introduced the mercurial manometer of U form, thus bringing the observation of blood-pressure within the compass of a reasonable space, and, in addition, made prolonged experiments possible for the first time, by retarding the coagulation of the blood in the tube leading from the artery. This he accomplished by filling the tube with a saturated solution of sodium carbonate, which is commonly employed to this day.

B. The Kymographion.—Ludwig's contribution was of even greater importance, because the graphic method, which he applied to the study of the blood-pressure in 1847,[3] has proved not alone a means of permanent and unimpeachable record, but an invaluable aid in the elucidation of all the mechanical problems of physiology. To his kymographion we owe nearly all our accurate knowledge of the circulation. This method of obtaining a blood-pressure tracing, by a recording manometer connected directly with an opened artery and writing upon a revolving cylinder, is so familiar as to need no explanation here.

---

[1] Hales, Stephen. Statical Essays: containing Hæmastatics. London, 1733, vol. ii, pp. 1 and following.

[2] Poiseuille, I. L. M. Recherches sur la force du cœur aortique. Paris, 1828, pp. 23 and following. Quoted by Tigerstedt, Lehrbuch d. Physiol. des Kreislaufes, Leipzig, 1893, p. 322.

[3] Ludwig, C. Beiträge zur Kenntnis des Einflusses der Respirationsbewegungen auf den Blutdruck im Aortensysteme. Arch. f. Anatomie, Physiologie u. Wissenschaftliche Med., etc., 1847, p. 261 and following.

That this method does not give an accurate record of all the events within the artery is not so well known, and is scarcely mentioned in the current text-books on physiology, because these confine themselves to the discussion of mean blood-pressure. The clinical methods for investigating blood-pressure, however, never determine the mean pressure, but either the maximum (systolic) or minimum (diastolic), and much confusion has arisen through a lack of appreciation of this fact. At the outset, therefore, everyone who would intelligently use these clinical instruments should understand the manometric blood-pressure trace and its limitations.

2. THE NORMAL MANOMETRIC TRACE

Such a tracing is shown in Fig. 1. Two distinct sets of fluctuations are apparent in it. A careful comparison with a simultaneous record of the movements of the chest would show that the larger and more gradual rises and falls correspond with expiration and inspiration respectively; the small and rapid oscillations are those due to systole and diastole of the ventricles of the heart. With a rapidly revolving cylinder these pulse-waves may be more easily followed; but this is not desirable for the determination of blood-pressure. It is evident from this tracing that the arterial blood-pressure is not a constant quantity, susceptible of easy measurement, even for a short period of time, but, under the conditions of this experiment, has a certain average level about which it fluctuates rhythmically. This average height of the curve above the base-line of atmospheric pressure may be easily calculated for any given period of time, and, when multiplied by 2,[1] gives an exact measure in mm. Hg. of the average or mean blood-pressure.

---

[1] In all accurate U-tube manometers the displacement of the surface of the mercury in the two arms is, of course, equal in amount, but opposite in direction; therefore the height of the column is twice as great as the movement in the open arm.

3. THE ERRORS OF THE MERCURIAL MANOMETER —COMPENSATED MANOMETER

So heavy a liquid as mercury cannot possibly follow the rapid cardiac and respiratory variations in pressure, and it is to be emphasized that the extent of these on the manometric trace affords no measure of their actual value. Even for the accurate determination of mean pressure, certain conditions are necessary, the most important of which is the calibre of the tube. v. Kries[1] found that a manometer of 4 mm. diameter gave readings which did not vary more than three per cent. from the actual mean pressure, when the pressure was subjected to rapid variation, as in blood-pressure experiments. He advocated a constriction of the tube leading from the

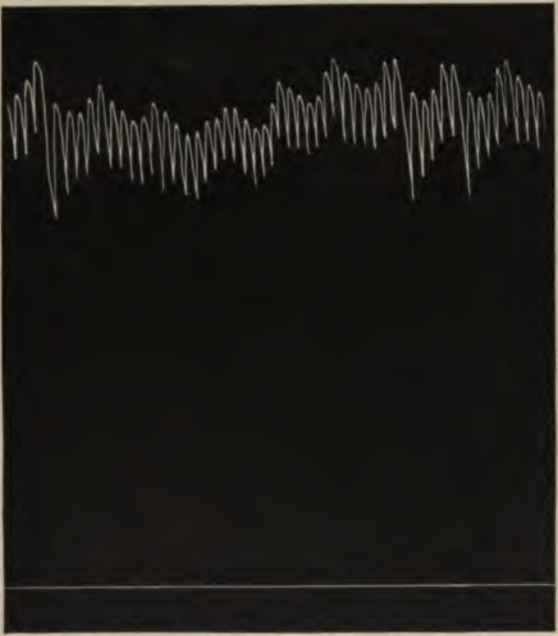


FIG. 1.—NORMAL MANOMETRIC TRACE.
From the carotid of a dog.

---

[1] v. Kries, Dr. Ueber die Bestimmung des Mitteldruckes durch das Quecksilbermanometer. Arch. f. Anatomie u. Physiologie, Physiologische Abtheilung, 1878, pp. 430 and following and p. 440.

artery, which almost obliterates the pulse wave in the manometer, and Marey[1] constricted the manometer itself at one point, calling his instrument a compensated one. Such instruments require five seconds to reach the true level, but then give exact readings of mean pressure.

4. ELASTIC MANOMETERS—MAXIMUM AND MINIMUM MANOMETERS

For most of the purposes of physiological and pharmacological experiment, a knowledge of the changes in mean blood-pressure has been considered sufficient. Attempts have been made, however, to study quantitatively the cardiac variations by means of elastic manometers of more or less complicated construction, such as are used to record intracardiac changes. Chaveau and Marey, Fick, Hürthle[2] and others have worked in this field, some using air and some water in transmitting the vibrations, and quite a controversial literature has grown up over the accuracy of the respective vehicles, so that none of their results can be considered as having absolute value. When controlled, however, by readings of maximum and minimum pressures during short periods, taken by means of mercurial manometers fitted with valves so as to record only maximum or minimum pressures, they probably give us a fair insight into the systolic and diastolic fluctuations of blood-pressure, and a criterion by which we may judge the validity of our clinical approximations.

These methods, all measuring directly the pressure within an opened artery, are obviously inapplicable to the study of the subject in man, or even in animals, except under certain fixed conditions. Their results, however, must always be the starting-point for any extension of our knowledge. For this reason, it seems to me necessary to consider some important features of the arterial circulation, as it has been elucidated under the simpler conditions of physiological investigation, before turning to the complex problems of the indirect estimation of human blood-pressure in health and disease.

---

[1] Marey. Annales des sciences naturelles. 4ᵉ série, Zoologie, vol. viii, 1858, p. 350. Quoted by Tigerstedt, p. 325.

[2] See Tigerstedt, R. Lehrbuch d. Physiol. des Kreislaufes, Leipzig, 1893, pp. 83–89.

CHAPTER II

BLOOD-PRESSURE IN THE NORMAL ANIMAL

1. General features of the arterial circulation.

A. The flow of liquid through tubes.

2. The factors which determine blood-pressure.

A. The energy of the heart.

- Influence on blood-pressure of volume output and of rate.
- Influence of blood-pressure on rate; the depressor mechanism; on volume output.
- Influence of intra-abdominal pressure on blood-pressure.

B. The peripheral resistance.

- The vaso-motor tone.
- Its presence in veins as well as arteries.
- Vaso-constrictor nerves and centres.
- Necessity of the vaso-motor tone for the maintenance of the circulation.
- The vaso-dilator fibres.
- Local and general variations in vaso-motor tone.
- The vascular reflexes.
- The depressor nerve.
- Pressor and depressor fibres.
- Importance of the splanchnic circulation.
- Relation between different vascular areas.

C. The elasticity of the arterial wall.

- The distensibility of the arteries and their resistance to strain.
- The variation of elasticity with internal pressure.

D. The volume of circulating blood.

- Smallness of the blood-volume contrasted with the capacity of the relaxed vascular system.
- Effect of great diminution in blood-volume on mean pressure only temporary.
- Insignificant effect of increased volume.
- Relation of systolic, diastolic, and mean pressure after hæmorrhage.

3. The mean aortic blood-pressure in different animals.

   A. Direct determinations of human blood-pressure.

4. The blood-pressure in different arteries.

5. The periodic variations of aortic blood-pressure.

   A. The cardiac variations. Systolic and diastolic pressures.
   B. The respiratory variations.
   C. The Traube-Hering waves.

6. Spontaneous non-rhythmical variations of aortic blood-pressure.

7. The influence of asphyxia on blood-pressure.

8. The influence of atmospheric pressure on blood-pressure.

9. The influence of muscular work on blood-pressure.

CHAPTER II

BLOOD-PRESSURE IN THE NORMAL ANIMAL

1. GENERAL FEATURES OF THE ARTERIAL CIRCULATION

At the centre of the circulation works the heart, pumping the blood from the veins into the great arteries, the ventricles first filling, then in a shorter time discharging their contents with great velocity; then resting to receive the next charge, and again to empty themselves, in rhythmic sequence. At the periphery, with quiet, even flow, the blood-stream in the vast capillary areas moves slowly toward the veins, everywhere flooding the tissues with nutriment and oxygen. From heart to capillaries the blood is carried by the arteries, continually branching from the largest trunks to the smallest arterioles, and within their walls this wonderful transformation of an intermittent jet into a continuous flow takes place. It is evident, then, that they are not mere conducting tubes, like so many water pipes, but that they exert a powerful influence upon the blood-current. For the better understanding of this function of the arteries, and of the causes of the arterial blood-pressure, we must turn to certain facts concerning the flow of liquid through tubes.

A. The Flow of Liquid through Tubes.—Since liquids are almost incompressible, it is clear that an intermittent flow through rigid tubes must always be the same at all points, and exactly the same amount must leave the tube as enters it. With distensible tubes of elastic material, such as the arterial walls, the conditions are different. If the tube be short and the calibre large in proportion to the size of the pump used, the walls may not be put appreciably on the stretch and an intermittent flow will occur, much as in a rigid tube. If, however, a larger tube be used and the open end narrowed to create a resistance to the outflow, with the first injections little fluid will leave the tube, the bulk accumulating within and distending it. This, of course, raises the pressure, which in turn increases the outflow, so that, as the experiment continues, the flow gradually increases until a steady stream is delivered, which just equals the amount pumped in during the same time. When this point is reached the system is in equilibrium, and the amount of distention of the tube and the pressure will bear an exact relation to the volume of intake and the size of the outlet.

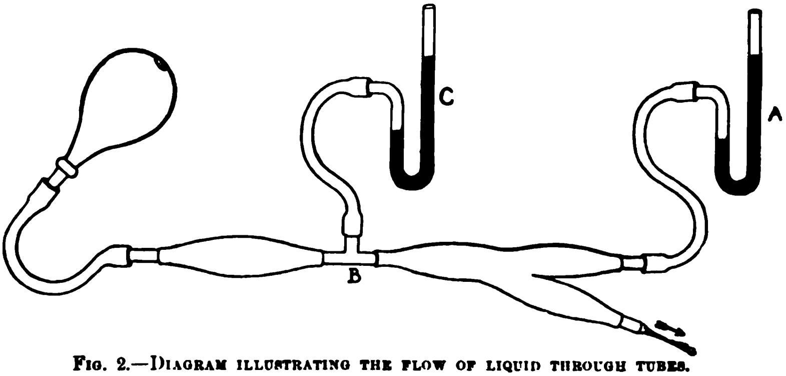


FIG. 2.—DIAGRAM ILLUSTRATING THE FLOW OF LIQUID THROUGH TUBES.

Such an experiment is shown in Fig. 2. If we connect a manometer (A) with the end of the tube we can measure the Total or End Pressure at that point. If, at some other point, a T-canula (B) be introduced without obstructing the flow and a manometer (C) connected with it, a pressure will be registered slightly lower than the end pressure. This is the Lateral Pressure, and is the force exerted on the walls of the tube at that point. The difference between it and the end pressure is due to the fact that the latter contains the pressure due to the velocity of the stream in addition. If the outlet be widened or the rate of the pump diminished, the lateral pressure will be seen to fall. Should this be continued progressively, a point will be reached at which the outflow will again become intermittent and lateral pressure will only be evident at each stroke of the pump.

These experiments prove that two conditions must be present to account for the disappearance of the pulse wave in the normal arterial circulation: they are, the elasticity of the arterial wall, and resistance to the flow, which we speak of as the peripheral resistance. They show in addition that, at any given point of the arterial system, the blood must exert a pressure against the arterial wall, which we may measure through a T-canula introduced into the artery, and a total pressure, which will equal the side pressure plus the velocity of the stream, and is recorded when we attach a manometer to a straight canula within the artery. As the common carotid artery leaves the aorta at practically a right angle, the end pressure within it gives the lateral pressure within the aorta.

As the scope of this book does not include the capillary and venous circulations, whenever subsequently blood-pressure is referred to, it is understood that arterial blood-pressure is meant.

2. THE FACTORS WHICH DETERMINE BLOOD-PRESSURE

In any individual, the blood-pressure at a given moment depends upon four separate factors: a. the energy of the heart; b. the peripheral resistance; c. the elasticity of the arterial walls; d. the volume of circulating blood. These may all vary under normal conditions, and in pathological states become greatly altered. Not only do they vary independently of one another, but so close is their connection, especially through the vaso-motor and cardiac nervous mechanisms, that changes in certain of them may produce marked secondary effects upon the others. For this reason the necessary conditions of experimental study are difficult and the results conflicting, so that our knowledge, even of the normal circulation, is incomplete, and, when it is applied to the still more complicated phenomena of disease, is most inadequate. Nevertheless, it is of the utmost importance that our clinical concepts and theories be built only upon the firm foundation of established fact, even though we might wish the foundation more ample.

A. The Energy of the Heart.—That the heart is the original source of all the forms of energy which we find manifested in the circulation is obvious. The blood-pressure necessarily depends in the first instance upon the force of ventricular systole.

Since the work of the heart depends upon the volume of blood discharged into the aorta, the velocity imparted to it, and the aortic pressure overcome, it is plain that a difference in blood-pressure may be brought about by change in either the volume output or the velocity of discharge.

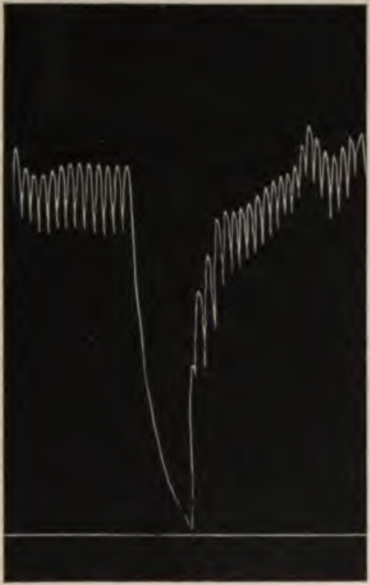


FIG. 3.—FALL IN BLOOD-PRESSURE PRODUCED BY STIMULATION OF THE PERIPHERAL CUT END OF THE VAGUS.

Trace from dog's carotid. Tetanizing current.

Any increase in the volume output of the ventricles causes a rise in pressure, and any diminution a fall, if the peripheral resistance remains unchanged. Such increase may be brought about in three ways: either by a larger systolic pulse-volume, or by a greater number of systoles per minute, or by both combined. The fall of blood-pressure which follows strong stimulation of the vagi peripherally is produced by both slowing the heart and decreasing the systolic output; and the two most commonly coincide. Such a fall in pressure, with marked decrease in ventricular output and slowing of the heart, is beautifully illustrated by a tracing of Porter's[1] from an experiment, in which the diminished cardiac energy followed tying off the circumflex branch of the coronary artery in a dog.

Rate and volume do not necessarily follow one another, however, for both Woolridge[2] and Pawlow,[3] by the stimulation of certain augmentor nerve branches in dogs, obtained increase in pulse volume and rise in pressure without change in frequency. This was probably due to greater suddenness of contraction, allowing a longer diastolic period for the filling of the ventricle. Vice-versa, increased rate does not always raise pressure. Münzel[1] demonstrated this most clearly. Having first severed the spinal cord to eliminate all vaso-motor influences, he stimulated the accelerator nerve and, in spite of the extreme rise in rate, the change in pressure was insignificant. For greater frequency to raise pressure there must be a large amount of blood in the great veins awaiting entrance to the heart, and a sufficient peripheral resistance. O. Frank[2] holds that, under normal conditions, pure rate variation (excluding any dynamic change in the heart) has such a relation to volume output, that the latter is greatest at a moderate rate, and hence, this gives the highest blood-pressure; that a change either way results in lower pressure; but within reasonable limits, difference in rate has slight effect on pressure.

---

[1] Porter, W. T. Further Researches on the Closure of the Coronary Arteries. Journal of Exper. Med., 1896, vol. i, p. 51; also, Amer. Text-Book of Physiol., second edition, 1900, vol. i, p. 182.

[2] Woolridge, Leonard. Ueber die Function der Kammernerven des Saügethierherzens. Arch. f. Anatomie u. Physiologie, Abtheilung f. Physiologie, 1883, p. 587.

[3] Pawlow, I. P. Ueber den Einfluss des Vagus auf die Arbeit der linken

The relation between cardiac energy and blood-pressure is a double one, however, and variations in the latter have a considerable influence on the heart. In the normal animal the pulse rate is slowed by raising arterial pressure, and hastened by lowering it, as Marey[3] first proved. Since this does not as a rule occur after division of the vagi, it is evidently due to action on the vagus centre, the more so, since the majority of observers agree that variations in pressure have, of themselves, little effect on the isolated heart.[4] This action is in part reflex, in part direct.

Herzkammer. Arch. f. Anatomie u. Physiologie, Abtheilung f. Physiologie, 1887, p. 452.

[1] Münzel, E. Pulsfolge und Blutdruck nach der Durchschneidung der Nervi Vagi. Arch. f. Anatomie u. Physiologie. Physiol. Abtheilung, 1887, p. 131.

[2] Frank, O. Einfluss der Haüfigkeit des Herzschlages auf den Blutdruck. Zeitschr. f. Biol., 1901, vol. xxiii, p. 1.

[3] Marey, I. Recherches sur le pouls au moyen d'un nouvel appareil enregistreur le Sphygmograph. Comptes Rendus des Séance et Mémoires de la Société de Biologie, 1859, p. 301 and following. Compare also, Loi qui préside à la fréquence des battements du cœur. Comptes Rendus Hebdomadaires des Séances de l'Academie des Sciences, 1861, vol. lviii, pp. 95–98. La circulation du sang à l'état physiologique et dans les maladies, Paris, 1881, pp. 334–340.

[4] Tigerstedt, Lehrbuch, pp. 295–298.

The reflex reaches the vagus centre by way of the most important centripetal nerve of the heart, the depressor nerve. This nerve, which exists as a separate anatomical structure in various warm-blooded animals, and whose homologue has been traced in man, has its endings in the ventricular wall and its central connections through the vagus. If it be divided, no change in heart action or blood-pressure normally follows;[1] and the same holds true if its peripheral end be stimulated. It is therefore wholly centripetal, and possesses no tonus. If, on the other hand, its central cut end be stimulated, a marked fall in blood-pressure and slowing of the heart follow. The former, as we shall see subsequently, is due to an inhibition of the vasomotor centre. The latter, since it never occurs after division of the vagi, must be dependent on stimulation of the centre for these nerves. In the depressor nerve, then, the heart possesses a protective mechanism of the first importance, by means of which it is able to shield itself automatically from the overwork entailed by continued high blood-pressure. Under ordinary life conditions the nerve is probably put in action through the stimulation of its endings by the abnormal tension of the ventricular wall, which high pressure causes.

The direct action of high arterial pressure upon the vagus centre was demonstrated by Franck,[2] who isolated the brain from the rest of the body of a dog, leaving only the vagi intact. Through the severed carotids he kept up an artificial circulation, and whenever he increased the pressure within them the heart-beat became slower. The same effect has been produced by increasing intracranial pressure through a trephine opening (Leyden),[3] and the fact has important clinical bearings.

The effect of the blood-pressure upon the volume output of the ventricle is a complicated one and observations on it do not show uniform results. The output depends largely upon the amount of blood brought to the heart by the great veins,[1] and in this way abdominal massage produces a rise in blood-pressure; but with a high peripheral resistance the heart will not empty itself of the increased volume and the rise will be absent.[2] Plethysmographic studies by Roy and Adami,[3] and others, show an increasing quantity of blood remaining in the ventricle after systole, at high pressure, even though the volume output may be increased. There is, therefore, a limit to the reserve force of the heart, so that, if the peripheral resistance be too much increased, the volume output of the ventricle will diminish. This, as well as the reflex depressor mechanism, set an upper limit to the blood-pressure, beyond which no increase in peripheral resistance or total blood-volume can augment it.

---

[1] Ludwig u. Cyon. Ber. d. sächsischen Gesellsch. d. Wiss., math. phys. Cl., 1866, p. 319. Quoted by Tigerstedt, Lehrbuch, p. 280.

[2] Franck. Traveaux du laboratoire de Marey, 1877, vol. iii, pp. 276 and following.

[3] Leyden, E. Beiträge und Untersuchungen zur Physiologie und Pathologie des Gehirns. Arch. f. pathol. Anat. u. Physiol. u. f. klin. Med., 1866, vol. xxxvii, p. 550.

Marked lowering of blood-pressure, especially of the degree obtained after section of the splanchnic nerves or spinal cord, by diminishing the amount of blood returned to the heart, has a considerable indirect effect on the cardiac energy. O. Frank[4] showed plainly in the isolated frog's heart, that when the filling of the ventricle is much decreased the energy of contraction is conspicuously less. This in turn will produce a still further fall in pressure, and hasten the death, which, we shall see later, follows complete vaso-motor paralysis.

In this connection, the effect on blood-pressure of increasing the intra-abdominal pressure may be mentioned. It has been investigated most recently by Quirin,[5] who, like previous observers, finds that, up to a certain point, increasing abdominal pressure raises the arterial pressure. Beyond this point it causes a fall in blood-pressure, which will finally lead to death of the animal, if compression of the abdomen be continued. The explanation of the primary rise is unquestionably the emptying of the contents of the abdominal veins into the right heart, followed by increasing resistance due to compression of the abdominal arteries. When this becomes too great, the heart, which at the same time receives a diminished amount of blood from the veins, exhausts its reserve force, and the blood-pressure rapidly falls until the heart ceases to beat.

---

[1] Howell and Donaldson. Philosophical Transactions, 1884, pp. 152–154. Quoted by Tigerstedt, Lehrbuch, p. 297.

[2] Tigerstedt, Robert. Studien über die Blutverteilung im Körper. Skandinavisches Arch. f. Physiol., 1891–92, vol. iii, pp. 207–219.

[3] Roy, Professor, and Adami, I. G. Remarks on Failure of the Heart from Overstrain. Brit. Med. Jour., 1888, vol. ii, pp. 321 and following.

[4] Frank, O. Zur Dynamik des Herzmuskels. Zeitschr. f. Biologie, 1895, vol. xxxii, p. 370, and Isometrie und Isotonie des Herzmuskels. Zeitschr. f. Biologie, 1901, vol. xli, p. 14.

[5] Quirin, Alexander. Ueber das Verhalten des normalen und pathologisch gesteigerten intraabdominalen Druckes und seine Rückwirkung auf die arterielle Blutcirculation. Deutsch. Arch. f. klin. Med., 1901, vol. lxxi, p. 79.

B. The Peripheral Resistance.—In the simple experiment with a distensible tube, it is plain that narrowing the outlet, other conditions remaining unchanged, causes a rise in pressure within the tube. The same holds equally for the arterial system. Increased resistance and higher pressure, diminished resistance and lower pressure, are invariable, unless some counterbalancing change in the force of the heart occurs.

Since normally there exists a considerable pressure within the arteries, there must be a well-marked resistance as its cause. A small part of this is due to friction, dependent on the viscosity of the blood and the continual branching of the arterial tree into smaller and smaller subdivisions. The really effectual cause, however, is the so-called tonus of the blood-vessels, the state of partial contraction which the unstriped muscle of their walls maintains continuously during health. From the standpoint of the blood-pressure and its variations, the vaso-motor mechanisms are by far the most important facts in circulatory physiology, and should be carefully studied by all who attempt the interpretation and clinical use of blood-pressure determinations. Our knowledge we owe to many investigators, beginning with Henle, Claude Bernard, Brown-Séquard and Schiff. Only the most important facts for the clinician, among the vast number which have been accumulated in this field, can be touched on here.

All blood-vessels which contain any muscle fibre possess tonus; that is, not only the arteries, but also the veins. This has been proved by Mall[1] for the portal vein. He compressed the aorta high up so as to cut off the abdominal circulation, and then, on stimulation of a splanchnic nerve, observed visible contraction of the portal vein, which emptied its contents into the liver. v. Bezold and Gscheidlen[1] noted the same for the general circulation, by stimulating the spinal cord after the heart had ceased to beat.

---

[1] Mall, F. Der Einfluss des Systems der Venæ portæ auf die Verteilung des Blutes. Arch. f. Anat. u. Physiol. Physiol. Abth., 1892, pp. 418 and following.

Under normal conditions the tone of the vessels depends on the balance between the opposing action of two sets of nerves, vaso-constrictor and vaso-dilator. The existence and course of these nerves has been demonstrated for practically every organ and portion of the body, the brain excepted.[2] The constrictor nerves are the more important for the general, if not for the local, circulation. Their tonus is derived in the first instance from the vaso-constrictor centre, situated in the upper part of the medulla oblongata. Section of the medulla below the centre is followed by dilatation of all the arteries of the body and great fall in blood-pressure. If the peripheral cut end be stimulated, a rise in pressure occurs, which may reach the extreme height of seven times the previously existing figure. That this is due to a general constriction of the peripheral arteries was made certain by Ludwig and Thiry,[3] and by Goltz,[4] who obtained the same rise after section of all the cardiac nerves; v. Bezold,[5] who first performed the experiment, having considered it due to accompanying increased heart-action.

This bulbar centre is not the only one, however, for, after section of the cervical cord, the arteries may in time recover their tone. Other centres are therefore present in the lower neurons. If, instead of simply cutting the cord, it be destroyed throughout its whole length, the vessels never recover their tone, the blood all collects in the veins, the heart-beat becomes progressively weaker, and death ensues.[1] (Compare page 15, this chapter.) Such an experiment proves conclusively that the vaso-motor tone is an absolute necessity for the maintenance of the circulation, that of the veins equally with the arteries; only by the existence of it is an adaptation of the total vascular stream-bed to the blood-volume possible. Its absence is followed by stagnation of the blood in the veins and eventual failure of the heart, because no blood is brought to it. It also demonstrates the existence, below the bulbar, of spinal vaso-motor centres, which are of the first importance. In the study of the local, not the general, circulation, lower centres have been found, which are very probably in the sympathetic ganglia. Their existence is evidenced by the return of tone in the vessels of the dog's legs a number of days after destruction of the lumbar cord, which contains the spinal centres for the lower extremity;[2] also by the gradual recovery of tone in isolated arteries all of whose vaso-motor nerves have been severed.[3] These peripheral centres are important as explaining the probable mechanism of the action of the vaso-dilator nerves, and for the vascular reflexes; but, as already said, they cannot suffice for the recovery of general vaso-motor tone throughout the body after destruction of the cord.

---

[1] v. Bezold, A., and Gscheidlen, Rich. Von der Locomotion des Blutes durch die glatten Muskeln der Gefässe. Untersuchungen aus dem physiol. Lab. in Würzburg, 1867, pp. 355 and following.

[2] Hill, Leonard. The Cerebral Circulation. London, 1896.

[3] Ludwig und Thiry. Sitzungsber. d. kais. Akad. d. Wissensch., math. naturw. Cl., 1864, vol. xlix, Abth. 2, pp. 421–454. Quoted by Tigerstedt, Lehrbuch, p. 542.

[4] Goltz, Fr. Ueber den Tonus der Gefässe und seine Bedeutung für die Blutbewegung. Arch. f. pathol. Anatomie u. Physiologie und f. klin. Med., 1864, vol. xxix, pp. 394–432.

[5] v. Bezold, Albert. Untersuchungen über die Innervation des Herzens, 1863, 2te Abt., pp. 219 and following.

The vaso-dilator nerve-fibres possess no tonus, and section of them causes no narrowing of the corresponding arteries. They have not, therefore, the vital significance of the vaso-constrictors. For the adjustment of the local circulation to the demands made upon it, especially in relation to the blood-supply of secreting glands, they are of great use. In this way their activity affects the general blood-pressure. Their first absolute demonstration was in the chorda tympani nerve by Claude Bernard,[1] and the extent of their influence may be judged from the fact that, in his experiment, their stimulation increased the blood stream in the vein leading from the submaxillary gland from 5 c.c. in sixty-five seconds to 5 c.c. in fifteen seconds, and the flow pulsated as from an open artery.

---

[1] Goltz, Fr. Ueber den Tonus der Gefässe und seine Bedeutung für die Blutbewegung. Arch. f. pathol. Anatomie u. Physiologie und f. klin. Med., 1864, vol. xxix, p. 422. Ueber die Functionen des Lendenmarks des Hundes. Arch. f. d. gesam. Physiol. d. Menschen u. d. Thiere, 1874, vol. viii, pp. 485 and following.

[2] Goltz, Fr., und Ewald, O. R. Der Hund mit verkürztem Rückenmark. Arch. f. d. gesam. Physiol., 1896, vol. lxiv, p. 397.

[3] Tigerstedt, Lehrbuch, p. 513.

Anatomically, constrictor and dilator fibres are frequently found in the same nerve, as, for instance, the splanchnic. When thus associated they are distinguished by their different reaction to electrical stimulation, the dilators being affected by currents of weaker strength, and shocks in slower rhythm; they also respond less quickly, but continue longer in action. The vaso-dilator centres are not thoroughly known, but exist at least in the spinal cord.

The vaso-motor tone in particular vascular areas varies constantly in response to local needs. This is a physiological necessity, since increased function of a part must always be accompanied by increased blood-flow. It can be observed most easily in muscles, and in glands, secretion being attended by a very great increase in the amount of blood passing through their vessels. This, of course, is provided by a dilatation of the arteries throughout the gland. Such a general dilatation of the small arteries in any vascular area causes a fall in lateral pressure in the main artery supplying it. If the area be of sufficient extent, and is not compensated for by vaso-constriction in other vascular districts, a fall in mean aortic blood-pressure follows. On the other hand, the total end pressure in the smaller arteries is increased by their dilatation, since less energy is lost in overcoming friction central to them, and, what is most important, when certain clinical methods are considered, the maximum systolic pressure rises markedly. That this must be so is obvious from Bernard's experiment, in which the pulse, ordinarily imperceptible in the smallest arteries, became evident even in the

---

[1] Bernard, Claude. Leçons sur les liquides de l'organisme. Paris, 1859, vol. i, pp. 310–311; vol. ii, p. 275. Quoted by Tigerstedt, Lehrbuch, pp. 494 and 502.

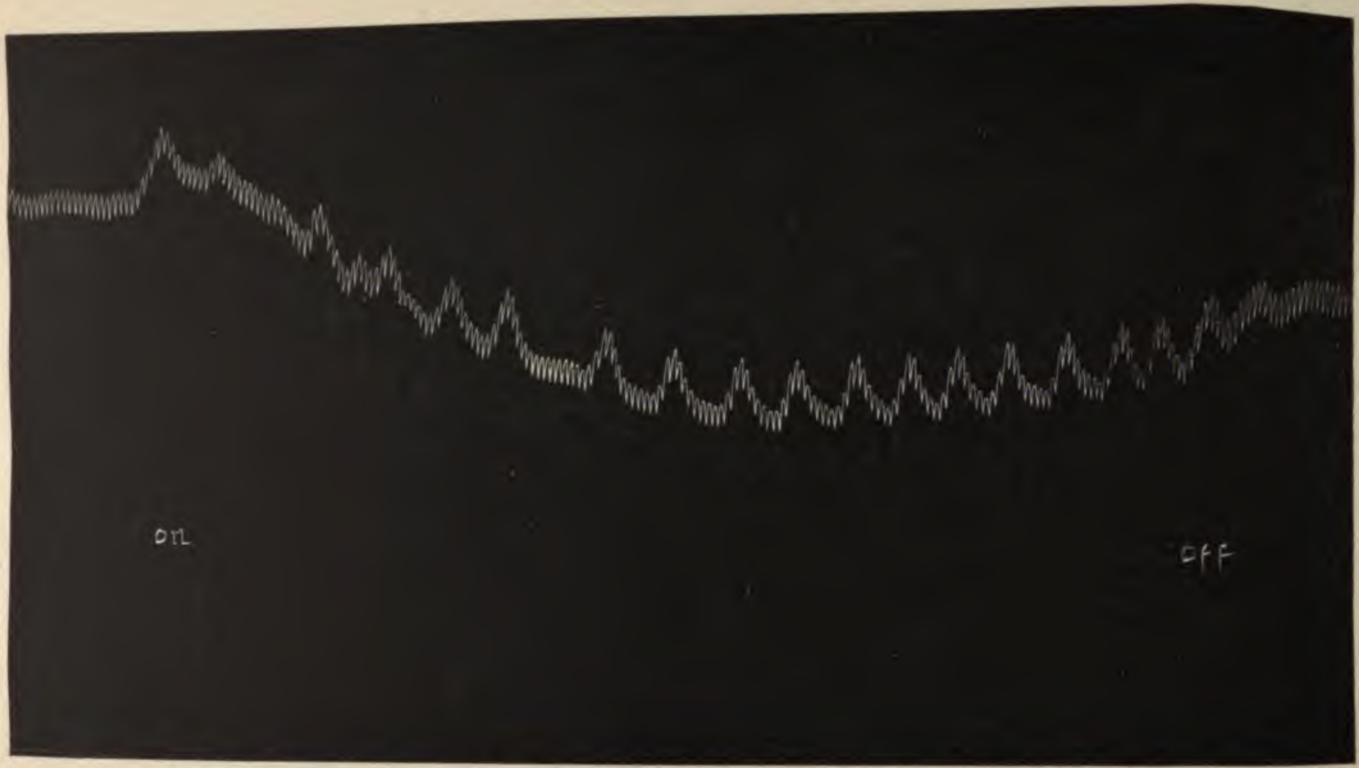


FIG. 4.—FALL IN BLOOD-PRESSURE PRODUCED BY STIMULATION OF THE CENTRAL CUT END OF THE VAGUS (depressor effect).
Trace from dog's carotid. Both vagi cut. Tetanizing current.

vein. The mechanism by which this variation in vascular tonus is brought about is a reflex one, either constrictor or dilator action being stimulated or inhibited, in response to a stimulus reaching the local, spinal, or bulbar centres along a centripetal nerve.

Vaso-motor tone, both local and general, is markedly affected by reflexes of remote origin. These vascular reflexes are of great significance for the general blood-pressure, as well as for the distribution of the blood, and have been extensively studied. They may be provoked by the stimulation of any afferent nerve. The only such nerve in the entire body, stimulation of which invariably lowers blood-pressure, is the depressor nerve. The fall, in this case, is due chiefly to a dilatation of the splanchnic vessels; but not wholly, for some lowering occurs after section of the splanchnic nerves.

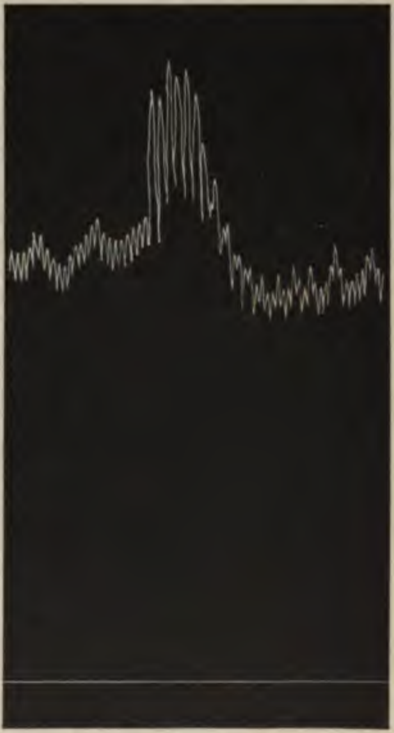


FIG. 5.—RISE IN BLOOD-PRESSURE PRODUCED BY STIMULATION OF THE ANTERIOR CRURAL NERVE (pressor effect).

Trace from dog's carotid. Tetanizing current.

With this single exception, stimulation of any centripetal nerve as a rule raises blood-pressure. This is easily shown on stimulation of the central cut end of the sciatic, anterior crural, or other large nerve trunk, and similar reflexes occur on irritation of parts of the cerebral cortex. The sensory nerves, however, contain some depressor fibres, for Howell[1] has shown that, if the sciatic is cooled to 0° C. central to the point of stimulation, a fall in pressure replaces the ordinary rise. Similarly, Hunt[1] found, that, in the early period of regeneration of the cut sciatic, only reflex vaso-dilatation follows stimulation, the reflex constriction being obtained later, when the nerve has been more completely repaired. Mechanical stimulation of the skeletal muscles, and of the mucous membrane of rectum and vagina, commonly results in a fall in pressure.[2] According to Porter,[3] this happens more readily when the animal is under ether, chloroform, or chloral, than under curare. This has a clinical bearing in relation to surgical shock, a condition in which depressor reflexes take the place of the normal pressor ones.

---

[1] Howell, Budgett, and Leonard. The Effect of Stimulation and of Changes in Temperature upon the Irritability and Conductivity of Nerve Fibres. Jour. of Physiol., 1894, vol. xvi, p. 298.

In reflex rises of pressure, the largest factor is vaso-constriction in the splanchnic area, though other vessels participate. With the exception of the brain vessels, in which vaso-motor changes have not been proven, the arteries of the muscles show the smallest reflex variation.

Of all the parts of the vascular system, the abdominal vessels, innervated by the splanchnic nerves, have by far the greatest effect on general blood-pressure. This is one of the most essential facts to bear in mind clinically. The extent to which section of the splanchnic nerves lowers the mean aortic pressure varies somewhat in different animals; in rabbits it almost equals section of the cervical spinal cord, while in the dog and cat it is less extreme. In all cases, however, and undoubtedly in man as well, the chief control of general blood-pressure is lodged here. One reason for this may be found in the size of this vascular area, which includes the portal vein and its branches, and is large enough to contain almost the whole blood-volume of the body. A second reason is the fact that of all the vaso-motor nerves, the splanchnics are the most easily affected by reflexes from any sensory nerve.[4]

The proof of their importance was furnished first by Ludwig and Cyon,[1] and by v. Bezold and Bever.[2] The former found that, after section of one nerve, the aortic pressure sank about 30 to 50 mm.; if the second were then cut, a further fall of 8 to 10 mm. was obtained. The latter observed practically the same lowering of pressure after section of both splanchnics in the thorax, as after division of the cord high up. When one considers that section of the cervical sympathetic, which innervates all the vessels of the head, exerts no material influence on aortic pressure, these facts become sufficiently striking. That the greater splanchnic nerves are not the sole vaso-motor supply for the abdominal organs, has been proved by Asp,[3] who found, after their division, a return of blood-pressure to its normal level in eleven or twelve days. This double innervation holds good for most arteries and in no wise detracts from the fundamental importance of the vessels of the abdominal viscera, as the main source of the normal peripheral resistance.

---

[1] Hunt. The Fall of Blood-Pressure Resulting from the Stimulation of Afferent Nerves. Jour. of Physiol., 1895, vol. xviii, p. 381.

[2] Tigerstedt, Lehrbuch, pp. 528, 529.

[3] Porter, W. T. Amer. Text-Book of Physiol., second edition, 1900, vol. i, p. 203.

[4] Tigerstedt, Lehrbuch, p. 520.

An equally striking demonstration that the intact splanchnic circulation can compensate for the most extreme narrowing of the rest of the arterial tree was furnished by the experiments of v. Bezold,[4] and of Ludwig and Thiry,[5] which have been frequently corroborated since. The former showed that, in an animal with severed cervical cord, ligature of the abdominal aorta itself, just below the renal arteries, had practically no effect on the carotid pressure, or on the frequency and force of the heart. The latter, under similar conditions, found a striking rise on ligature of the aorta above the diaphragm, that is, above the origin of the splanchnic vessels. These observations are of fundamental importance in the interpretation of the effects of arterio-sclerosis on the heart.

---

[1] Ludwig und Cyon. Bericht der sächsischen Gesellsch. der Wissenschaften, math. phys. Cl., 1866. p. 315. Quoted by Tigerstedt, Lehrbuch, p. 488.

[2] Bever, Carl, und v. Bezold, A. Von den Wirkungen der Nervi splanchnichi auf den Blutdruck im Aortensysteme. Untersuch. aus dem physiolog. Lab. in Würzburg, vol. i, 1867, pp. 314–325.

[3] Asp. Bericht der säch. Gesellsch. der Wissensch., math. phys. Cl., 1867, pp. 141, 142. Quoted by Tigerstedt, Lehrbuch, p. 491.

[4] v. Bezold, A. Untersuchungen über die Innervation des Herzens, Leipzig, 1863, pp. 223–229.

[5] Ludwig und Thiry. Sitzungsber. d. kais. Akad. d. Wiss., math. naturw. Cl., 1864, vol. xlix, 2, p. 442.

Finally, it must be remembered that, in reflex raising or lowering of blood-pressure, all the arteries of the body are not contracted or dilated at the same time. In general there seems to be an antagonism between the vessels of the internal organs and those of the skin and muscles, so that the one set will be more or less widened when the other becomes narrowed. This, however, is by no means invariable, and, under certain circumstances, both may be constricted together.[1]

C. The Elasticity of the Arterial Wall.—Were it not for the distensibility of the arteries, the work of the heart would be many times greater than it is. Each systole would then have to propel the total blood-column, and, during the period of active ventricular contraction, displace into the capillaries an amount equal to that injected into the aorta. The systolic blood-pressure would then be extreme, the diastolic practically zero, and the flow intermittent. The elastic arterial wall is a mechanical device of great importance. In it during systole a large part of the energy of the ventricles is made potential, to be utilized in moving the blood during diastole, thus converting an intermittent into a continuous propelling force, and distributing the heart's work over more than twice the time of its actual muscular contraction.

The elasticity of the arteries is very perfect; that is, they may be much distended by pressure and subsequently resume their original size. In the terminology of physics they possess wide limits of elasticity, as does india-rubber. It is this property of elastic distensibility which is important. They also possess coefficients of elasticity, which are more than ample to enable them to endure, without breach of continuity, any pressure they may ever be subjected to during life. Gréhant and Quinquard[2] found that the carotid of a dog could stand a pressure twenty times greater than the normal blood-pressure without tearing. For the human carotid the lowest pressure at which rupture occurred was 1.29 m. of mercury, at least eight times ordinary carotid pressure. It is therefore safe to assume that a healthy artery cannot be torn by any increase in blood-pressure which is possible during life.

---

[1] Tigerstedt, Lehrbuch, p. 529.

[2] Gréhant, N., et Quinquard, H. Mesure de la pression nécessaire pour determiner la rupture des vaisseaux sanguins. Jour. d. l'Anat. et de la Physiol. normal et pathol. de l'homme et des animaux, 1885, vol. xxi, pp. 287, 297.

Even under physiological conditions, however, the limits of elasticity of the arteries are not constant, but vary with the internal tension to which they are subjected. The exact manner in which the change occurs is somewhat in dispute, Roy's,[1] and Zwaardemaker's[2] experiments having shown the greatest elasticity at about the lower limit of normal blood-pressure for the animal, the elasticity decreasing at higher and lower pressures; Marey,[3] and Thoma and Kaefer,[4] on the other hand, finding the maximum in the unstretched artery, and a progressive loss of elasticity the higher the absolute pressure was raised. At least one fact is secure; at a high arterial pressure the distensibility of the arteries is distinctly diminished, and any increase in the systolic pulse-volume must raise the blood-pressure far more than a corresponding increase at lower pressures. Not only is this true as regards mean blood-pressure, but still more so when we consider systolic pressure, as will be seen in the study of certain morbid states. That, under these circumstances in the normal animal, the ventricle does not empty itself completely is probable; but in certain pathological conditions, in which high tension and a hypertrophied ventricle coincide, this diminishing arterial distensibility, and consequent increase in the systolic elevation of pressure, should be borne in mind.

D. The Volume of Circulating Blood.—The most conspicuous fact about the total blood-volume is its smallness, when compared to the full cubic capacity of the arteries, capillaries, and veins combined. This disproportion is compensated for, in the normal animal, by the continuous contraction of the smaller arteries, which we have studied as the vaso-motor tone. Hence, as we have seen, when this tonus is wholly abolished by destruction of the spinal cord, the blood-volume is insufficient to fill all the vessels, and is rapidly forced out of the arteries into the veins until death ensues, the animal practically bleeding to death into its own veins.

---

[1] Roy, Charles S. The Elastic Properties of the Arterial Wall. Jour. of Physiol., 1881, vol. iii, p. 141.

[2] Zwaardemaker, Dr. Over de Nitzetting der Slagaderen voor den Bloedsdruk. Nederlandsch Tijdschrift voor Geneeskunde, Tweede Reeks, 1888, vol. xxiv, i, pp. 61–78.

[3] Marey. Travaux du laboratoire de M. Marey, 1880, vol. iv, pp. 178 and following.

[4] Thoma, R., und Kaefer, N. Ueber die Elasticität gesunder und kranker Arterien. Arch. f. pathol. Anat. u. Physiol. u. f. klin. Med., 1889, vol. cxvi, pp. 9 and following.

As a factor in the control of the blood-pressure it is of less importance than the foregoing, chiefly because its variations are so easily compensated for. That death from hæmorrhage is attended by a fall in the blood-pressure is obvious. Under normal conditions, however, the abstraction of considerable amounts of blood causes a temporary fall in pressure, soon followed by a rise to near the former point. Tappeiner[1] found that one-fifth of the total blood-volume could be lost, and, within a short time, the pressure again reach a height sufficient to support life. The chief mechanism by which this is brought about is, of course, the increase of peripheral resistance through vaso-constriction. It is well known, though, that after severe hæmorrhage the kidneys secrete little urine, and Langley[2] has shown the same effect upon the salivary glands. It is also possible that the tissues return more fluid, and the liver more of its blood, to the general circulation. The heart's rate increases greatly with loss of blood, and it may be supposed to empty itself as completely as possible, so that really great losses (up to two-fifths of the total blood-volume) may affect mean blood-pressure surprisingly little.

On the other hand, Worm-Müller[3] has shown that an amount of fluid greater than the total blood-volume of the body can be transfused into the vessels, without increasing the blood-pressure above a point which it frequently reaches under ordinary conditions. Pawlow,[4] in the same way, was able to feed a dog enormous quantities of bouillon without any rise in pressure. In such experiments, part of the excess volume is removed from the circulation by the secreting organs, especially the kidney, and part by increased transudation into the lymph spaces and serous cavities ; the remainder is accommodated by the vessels, which dilate, either through a direct vascular reflex, or in response to a reflex from the heart by the depressor nerve. Under certain circumstances, such as too rapid infusion, the heart may empty itself less completely (Johannson and Tigerstedt).[1]

---

[1] Tappeiner. Bericht der sächs. Gesellsch. der Wissensch., math. phys. Classe, 1872, pp. 199 and following. Quoted by Tigerstedt, Lehrbuch, p. 345.

[2] Langley. Philosophical Transactions, 1889, vol. clxxx B, p. 128. Quoted by Tigerstedt, Lehrbuch, p. 350.

[3] Worm-Müller. Bericht der säch. Gesellsch. der Wissensch., math. phys. Classe, 1873, pp. 573–664. Quoted by Tigerstedt, Lehrbuch, p. 345.

[4] Pawlow, Joh. Zur Lehre über die Innervation der Blutbahn. Arch. f. d. gesam. Physiologie d. Menschen u. d. Thiere, 1879, vol. xx, pp. 23, 24.

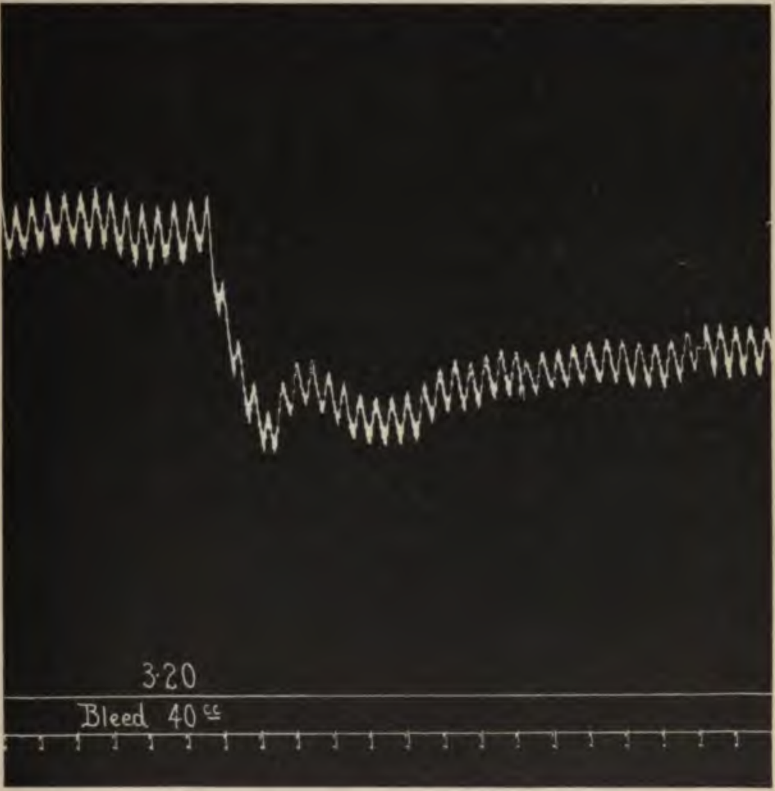


Fig. 6.—Fall in blood-pressure produced by hæmorrhage.
Trace from rabbit's carotid. Slow drum.

---

[1] Johannson, J. E., und Tigerstedt, Robert. Ueber die gegenseitigen Beziehungen des Herzens und der Gefässe. Skandinavisches Arch. f. Physiol., 1889, vol. i, p. 397.

Therefore, within certain wide limits, the volume of circulating blood has, in the normal animal, only a subordinate and temporary influence on mean blood-pressure.

It is evident that the regulating influences, which prevent excessive increase of the quantity of circulating fluid, will not be operative when that quantity has been abnormally reduced. Thus the transfusion of blood, or the infusion of normal saline solution into the veins, after profuse hæmorrhage, brings about a prompt rise in pressure. Dawson[1] has shown recently that, if the solution contain 0.25 per cent. sodium bicarbonate in addition to 0.8 per cent. of chloride, both the systolic and diastolic pressures will be higher than when saline alone is used.

Since our clinical methods measure systolic or diastolic, but never mean pressure, we must bear in mind that the rise in frequency of the heart-beat, which is one of the greatest compensating factors after hæmorrhage, must be attended with diminished systolic pulse-volume. This necessarily leads to decrease in systolic and diastolic variation in pressure. If the mean pressure remains unchanged, systolic pressure must fall and diastolic rise. Experiment XII, in the work of Howell and Brush, given on page 34 of this chapter, demonstrates this. A fall in mean blood-pressure during hæmorrhage will not be accurately measured by the change in systolic or diastolic pressure, but will be magnified by the former and minimized by the latter.

3. THE MEAN AORTIC BLOOD-PRESSURE IN DIFFERENT ANIMALS

No fact regarding the blood-pressure is better established than its wide range of variation in any individual. It is therefore impossible to speak of a normal value for blood-pressure, but only of certain normal upper and lower limits. In consequence it is difficult to compare the values found in different species, except as rough averages. There have been few studies on this question, but the following table, copied from Volkmann,[1] illustrates the usual figures sufficiently well.

---

[1] Dawson, Percy M. Effect of Intravenous Infusion of Sodium Bicarbonate after Severe Hæmorrhage. Am. Journ. of Physiol., 1904, vol. x, Proceed. of Am. Physiol. Soc., p. xxxvi.

Animal Species.	Blood-Pressure in mm. of Mercury.
Horse	321
Horse	214
Sheep	206
Calf	177
Large Dog	172
Sheep	169
Dog	166
Calf	165
Dog	157
Sheep	156
Calf	153
Horse	150
Cat	150
Dog	143
Goat (female)	135
Calf	133
Large Dog	123
Horse, old and thin	122
Small Goat (male)	118
Horse, old	110
Rabbit	108
Dog, young	104
Sheep, old	98
Rabbit	90

Volkmann concluded from these that the size of the animal does not determine the height of the blood-pressure, a decision which seems only partially justified, the horse, except for two old animals, showing only in the upper half of the list, and the rabbit among the lowest figures. If we consider the animals of medium size, leaving out the horse and rabbit, we find the figures lying between 98 mm. and 206 mm., readings which we shall see later are not very far from those found clinically in man.

As regards the height of blood-pressure in different individuals of the same animal species, almost as great variation seems to obtain. Crile[2] recorded the mean carotid pressure, under full anæsthesia, in 100 dogs. The highest was 170 mm., the lowest 80 mm., the average 125 mm. There was no relation between blood-pressure and body-weight.

A. Direct Determinations of Human Blood-Pressure.—A few observations of this kind have been made in the course of amputation, the pressures found in the femoral and brachial arteries varying between 100 mm. and 160 mm.[3, 4] These experiments are questionable from the ethical standpoint, and the results are useful merely as showing certain possible real values for human arterial pressure, not in any way its range.

---

[1] Die Hämodynamik, 1850, pp. 177, 178.

[2] Crile, Geo. W. Blood-Pressure in Surgery, Phila., 1903, p. 359.

[3] Faivre, I. Études Experimentales sur les Lésions Organiques du Cœur. Gaz. Méd. d. Paris, 1856, p. 727.

[4] Albert E. Einige kymographische Messungen am Menschen. Med. Jahrbücher. Wien, 1883, p. 254.

4. THE BLOOD-PRESSURE IN DIFFERENT ARTERIES

Since the various clinical methods for the estimation of blood-pressure deal with arteries as different in calibre and in their distance from the heart, as the brachial, radial, digital, temporal, and femoral, it is important that we should know what changes in pressure occur, as the blood travels from the aorta to the periphery. Those unfamiliar with the experimental facts might suppose that there is a continuous and more or less even fall in pressure from the largest to the smallest arteries. Poiseuille,[1] however, after his first researches, came to the opposite opinion, and thought that the mean pressure was equal in the various arteries and independent of their distance from the heart. This is not strictly true, but the striking fact has been established by all subsequent investigation, that the lateral pressure in the arteries, of not too small diameter, diminishes extremely slowly with increasing distance from the heart. v. Schulten,[2] for instance, found that the maximum pressure in the ophthalmic artery was only 2 to 15 mm. below that in the aorta. Some experiments of Hürthle's[3] with an elastic manometer, which are quoted later (see page 33), are equally demonstrative of this point.

In his tables the constancy of diastolic pressure in carotid, renal, and femoral arteries is striking. Recent experiments by Dawson,[4] as yet unpublished, showed, for the dog, the greatest uniformity in minimum pressure throughout all the larger arteries. The work of Tschnewsky,[1] also, though not undertaken with reference to this particular point, showed in seven dogs an average mean pressure in the femoral of 77 mm.; in eight dogs of similar size the average mean pressure in the common carotid was 92.6 mm.

---

[1] Poiseuille, J. L. D. Recherches sur la Force du Coeur Aortique. Thesis, Paris, 1828, pp. 31 and following. Quoted by Tigerstedt, Lehrbuch, p. 351.

[2] v. Schulten, M. W. Untersuchungen über den Hirndruck, mit besonderer Rücksicht auf seine Einwirkung auf die Circulationsverhältnisse des Auges. Arch. f. klin. Chir., 1885, vol. xxiii, p. 463.

[3] Hürthle, Karl. Ueber den Ursprungsort der sekundären Wellen der Pulscurve. Arch. f. d. gesam. Physiologie d. Menschen u. d. Thiere, 1890, vol. xlvii, pp. 32–34.

[4] Dawson, Quoted by Erlanger. Am. Journ. of Physiol., 1904, vol. x. Proceed. of Am. Physiol. Soc., p. xiv. (Experiments to be published in full later.)

Under certain conditions, such as external cold, peripheral arteries like the digital may become much contracted, and the pulse-wave in them be lost nearer the heart than usual. Under such circumstances the lateral pressure in them will fall more rapidly than normal, especially the systolic pressure. In general, though, it may be accepted that no marked fall occurs within the arterial system until the smallest arteries and arterioles are reached, for these are the seat of most of the peripheral resistance. Therefore, in the larger systemic arteries used for the clinical estimation of blood-pressure, the lateral pressure, especially the diastolic, closely approximates that within the aorta.

In the lesser circulation, as is well known, much lower pressures obtain, since this is true for the right ventricle as compared with the left. The direct estimation of blood-pressure in the pulmonary artery is very difficult, without producing markedly abnormal conditions. The best experiments by Bentner,[2] Lichtheim,[3] Bradford and Dean,[4] and Knoll,[5] have shown values, as compared with aortic pressure, between 1:2.6 and 1:13.4. These were in the rabbit, dog, and cat. In all probability it does not average more than one-fifth the height of mean aortic pressure.

---

[1] Tschnewsky, J. A. Ueber Druck, Geschwindigkeit und Widerstand in Strombahn der Arteria Carotis und Cruralis, etc. Arch. f. d. gesam. Physiol., 1903, vol. xcvii, pp. 262 and 270.

[2] Bentner, A. Ueber die Strom- und Druckkräfte des Blutes in der Arteria und Vena pulmonalis. Zeitschr. f. rat. Med., 1852, vol. ii, pp. 106, 120.

[3] Lichtheim, Ludwig. Die Störungen des Lungen Kreislaufes und ihr Einfluss auf den Blutdruck. Breslau, 1876, p. 31. Quoted by Tigerstedt, Lehrbuch, p. 444.

[4] Bradford, I. R., and Dean, H. P. The Innervation of the Pulmonary Vessels. Proceedings of the Royal Soc. of London, 1889, vol. xlv, p. 370.

[5] Knoll, Philipp. Der Blutdruck in der Arteria Pulmonalis bei Kaninchen und seine respiratorischen Schwankungen. Sitzungsber. der kais. Akad. der Wissensch., math. naturw. Cl., 1888, vol. xcvii, Abth. iii, pp. 212, 214.

The most recent work on this subject, by Tigerstedt,[1] has brought out a number of facts of clinical interest. He finds, for the rabbit, that the maximum pressure in the right ventricle shows in general, when large pressure variations within the thorax are excluded, only slight fluctuation, within 10 mm. From individual to individual it differed somewhat, but lay between 11 and 25 mm. Even the great narrowing of the cross-section of the pulmonary circulation, which was produced by opening the left chest and tying off the root of the left lung, affected the maximum pressure in the right ventricle little or none, and caused no fall of mean aortic pressure. One-sided pneumothorax is therefore insufficient to raise the work of the right heart, and the mechanical obstacle to the lesser circulation in pneumonia amounts to nothing. He proves further, "that no definite relation can be discovered between the mean pressure in the greater circulation and the maximum pressure in the right ventricle. On the contrary the former can vary within very wide limits without any corresponding change in the other."

5. THE PERIODIC VARIATIONS OF AORTIC BLOOD-PRESSURE

As we saw in examining the normal manometric tracing (Fig. 1), the blood-pressure is undergoing constant oscillation above and below a certain average level. Two sets of these fluctuations may be clearly seen in the trace, the more rapid cardiac, and the larger respiratory waves. In addition, certain longer but slighter rises and falls of the whole curve may be indistinctly made out, the so-called Traube-Hering waves. These and certain other variations, which are of regular occurrence in physiological experiment, are of great significance in the clinical study of blood-pressure and should always be in mind. They will be considered in the order of their importance.

A. The Cardiac Variations—Systolic and Diastolic Pressures.—Every physician who feels the pulse must appreciate that, at the moment the pulse-wave strikes the finger, the pressure within the artery is raised considerably, falling again after the wave has passed. In the peripheral arteries this occurs an appreciable time after the systole of the ventricles, but in the aorta the maximum and minimum of pressure correspond exactly with systole and diastole of the heart. We may then speak of the highest and lowest pressure in the course of the pulse-wave as systolic and diastolic pressure, remembering that, the farther we go from the heart, the less synchronous these are with the events in the cardiac cycle which produce them. Since all of the indirect methods for determining blood-pressure clinically give estimations, more or less accurate, of either systolic or diastolic, but never of mean pressure, it is imperative that we should know as much as possible of the absolute amount of variation within one cardiac cycle.

---

[1] Tigerstedt, R. Ueber den Lungenkreislauf. Skandinav. Arch. f. Physiol., 1903, vol. xiv, pp. 267 and 285.

Unfortunately, as we have seen, the mercurial manometer gives no accurate measure of such rapid fluctuations. For this purpose the various forms of elastic manometer have been employed, none of them absolutely free from the possibility of error due to their own vibrations, but giving valuable results. Very few reliable data are available on this question. Hürthle,[1] in the dog, investigated the relation of systolic and diastolic pressure in different arteries, giving several tables, one of which follows:

PRESSURE VARIATION DURING A PULSE-WAVE IN

ARTERY.	CAROTID.	RENAL.	FEMORAL.
	75–160	72–170	78–240
	78–158	75–165	80–235
	76–160	72–170	80-244
	80–156	80–164	90–225
	96–166	96–178	100–228
	94–166	96–178	100–230

The pressure variations here recorded are the largest that have been observed, averaging over 100 per cent of the diastolic pressure. Especially striking is the higher systolic pressure in the femoral than in the carotid artery, which Tigerstedt[1] considers must be due either to error in the manometer, or to the addition, in the femoral, of a primary positive reflected wave to the pulse-wave. Howell and Brush,[2] in some recent work with maximum and minimum manometers, were unable to find such extreme differences, except after section of both

---

[1] Hürthle, K. Ueber den Ursprungsort der sekundären Wellen der Pulscurve. Arch. f. d. ges. Physiol., 1890, vol. xlvii, pp. 32–34.

OBSERVATIONS.	Diastolic pressure.	Systolic pressure.	Mean pressure calculated.	Mean pressure directly determined by mercury manometer.	Rate per minute.	Amplitude.	
Exp. X.	Mm.	Mm.	Mm.	Mm.		Mm.	
I.......	133	159	146	142.75	96	2.4	
II[1].....	132	167	149.5	144.92	96	1.75	[1] Severed both vagi.
III.....	174	201.5	187.75	183.87	210	2	
IV	172	199	185.5	182	210	2	
Exp. XI.							
I.......	100	156	128	127.49	114	9.83	
II[2].....	108	156	132	129.2	120	8.4	[2] Severed both vagi.
III.....	161	196	178.5	177.4	153	5.6	
IV	165	215	191.5	185.46	132	6.9	
V......	133	160	146.5	141.69	132	4.81	
VI	124	158.5	141.25	145.45	126	4.85	
Exp. XII.							
I.......	112.5	158.5	135.5	133.35	87	17.35	
II......	111	159	135	133.9	84	17.7	
III.....	110	157	133.5	133.77	84	16.87	
IV	112	155	135	136	82	13.40	
V[3].....	113	155	134	138.03	84	17.17	[3] Severed vagi and bled profusely.
VI	129	140	134.5	134.5	234	1.25	
VII.....	128	140.5	134.25	136	234	1.25	
Exp. XIII.							
I.......	143	226	184.5	182.86	153	8.5	
II[4]. ...	144	216	178	169	162	8.5	[4] Severed both vagi.
III	226	272	249	232.5	174	10	
IV[5].....	188.5	258	223.25	226	171	10	[5] Severed left splanchnic.
V[6].....	101	137	119	124.75	162	5.25	
VI	37	68.25	52.62	47.33	162	3.7	[6] Severed right splanchnic.
VII.....	36	70	53	51.75	162	3.7	
VIII....	35	71	53.25	53	162	4	
IX	38.5	74	56.25	50.82	162	4	
X......	41	79	60	52.82	162	3.9	
XI......	45.5	78	61.75	57.24	162	3	

---

[1] Tigerstedt, Lehrbuch, p. 352.

[2] Howell, W. H., and Brush, C. E., Jr.- A Critical Note upon Clinical Methods of measuring Blood-pressure. Boston Med. and Surg. Jour., 1901, vol. cxlv, p. 146.

splanchnics. Under normal conditions, twenty observations on dogs gave

Greatest difference	58 per cent. of the diastolic pressure.
Smallest difference	17 per cent. of the diastolic pressure.
Average difference	33 per cent. of the diastolic pressure.

In man, with his slower heart rate, they believe the difference must be greater. They also tabulate (see page 34) some interesting figures on the relation of systolic, diastolic, and mean pressures. From these they draw the following conclusions:

"1. The mean blood-pressure, as usually determined by means of the mercury manometer, corresponds with fair accuracy to the arithmetical mean of the maximum and minimum pressures."

"2. A rise of blood-pressure occasioned by an increased heart beat (section of both vagi) affects the diastolic pressure to a greater extent than the systolic pressure."

"3. A fall of blood-pressure occasioned by vascular dilatation (section of both splanchnics) affects systolic and diastolic pressures equally."

"4. It is possible for the systolic and diastolic pressures to be affected in opposite directions, as seen in Exp. XII, in which section of both vagi was intentionally accompanied by profuse hæmorrhage. In this case the mean pressure remained unchanged, the diastolic pressure rising 16 mm. and the systolic pressure falling 15 mm."

They believe, even for physiological purposes, that a record of systolic and diastolic pressure would be more instructive than the ordinary mean pressure. They think their experiments indicate, so far as they go, that even under extreme conditions of cardiac and vascular change, unless accompanied by hæmorrhage, the general trend of arterial pressure is shown either in systolic or diastolic pressure.

These problems, in the light of their great importance to clinical sphygmomanometry, deserve careful study. The various attempts to solve them by clinical methods I will not consider here, for their results have not absolute value.

B. The Respiratory Variations.—The relationship of aortic blood-pressure to the inspiratory and expiratory acts is too complex for full discussion, and is totally different in the normal animal and in one in which the thorax has been opened.

In the latter, the varying calibre of the smaller pulmonary vessels is the only factor concerned, and the blood-pressure is highest in the first half of inspiration and lowest in the first half of expiration, because the largest and smallest amounts of blood are brought to the left ventricle at these times. In the healthy animal, however, breathing spontaneously, there is added to this the aspirating force of inspiration for the blood in the great veins; the changes in the elastic pull of the lungs upon the heart, making systole more difficult and facilitating diastole, and upon the great vessels, alternately widening and narrowing them; and the changes in intra-abdominal pressure, influencing the blood-flow to the right heart. Besides all these, there are rhythmical variations in the activity of the vagus and vaso-motor centres in the medulla, synchronous with, and in some way related to, the activity of the respiratory centre. Both show a greater tonus during expiration, the heart rate becoming slower and the peripheral resistance higher; thus their effect upon blood-pressure is antagonistic.

The result of the interaction of these various conditions, some tending to raise and some to lower pressure, depends upon the depth and rapidity of respiration. According to Tigerstedt,[1] during quiet, superficial breathing the blood-pressure shows no variations. With more forcible and rapid breathing the pressure rises during expiration and falls during inspiration; while with deeper and slower respiration the maximum pressure coincides with the beginning of expiration, the minimum with the beginning of inspiration. In the latter case the pulse frequency is also increased during inspiration. This summary at once suggests its most important clinical application, which is to make all pressure estimations during quiet, unforced breathing.

C. The Traube-Hering Waves.—This name is given to certain periodic rises and falls in blood-pressure, which embrace several respiratory periods. They were first noted by Traube[2] and are evident in all prolonged pressure observations. According to Knoll[1] they correspond, as a rule, with similar rhythmical variations in the depth of respiration, and are therefore, in all probability, due to periodic changes in the tonus of the vaso-motor and respiratory centres. They must be borne in mind in all clinical observations, since changes of several millimeters in pressure, at the least, may be due to them.

---

[1] Tigerstedt, Lehrbuch, p. 462.

[2] Traube, L. Ueber periodische Thätigkeitsäusserungen des vasomotorischen und Hemmungs-Nervensystems. Centralblatt f. d. med. Wissensch., 1865, vol. iii, p. 882.

6. SPONTANEOUS NON-RHYTHMICAL VARIATIONS OF AORTIC BLOOD-PRESSURE

In many experiments, changes in pressure of moderate extent are noted from time to time without assignable cause. As we have seen, the vaso-motor centre responds by a vascular reflex upon stimulation of any sensory nerve, and these apparently spontaneous variations in blood-pressure may be reflex, or they may be due to changes within the central nervous system. One must be warned of their occurrence, as of the Traube-Hering waves, in order that they may not be interpreted as proving the success or failure of therapeutic procedures.

7. THE INFLUENCE OF ASPHYXIA ON BLOOD-PRESSURE

If an animal be deprived of oxygen, phenomena ensue which produce the picture we call asphyxia. If the deprivation be sudden, as on tying off the trachea, death occurs inside of five minutes and is preceded by convulsions. Where suffocation is more gradual, as in a closed chamber, the convulsions do not appear and life is more prolonged. In addition to the motor and respiratory manifestations of dyspnœa, however, there is a striking effect upon the vaso-motor and vagus centres. In the first stage, during which the respiratory centre is stimulated and the breathing is rapid and deep, the vaso-motor centre in the medulla is also thrown into action and a marked rise in blood-pressure occurs. This is accompanied by increasing slowness of the pulse from simultaneous activity of the vagus centre. In the later stages, as the irritability of the respiratory centre becomes exhausted, the other bulbar centres likewise fail; then the spinal vaso-motor centres begin to act and the blood-pressure is maintained at a high level, with increasing heart-rate and spasmodic, ineffectual respiratory acts, only to fall during the last seconds of life, when the spinal centres have become utterly exhausted.[1] This extreme rise of blood-pressure caused by asphyxia and, in a lesser degree, by slighter grades of deficient oxygenation of the blood, is of distinct clinical import, as in the study of arterial pressure in cardiac and lung conditions, laryngeal diphtheria, etc.

---

[1] Knoll, P. Ueber periodische Athmungs und Blutdruckschwankungen. Sitzungsber. d. kais. Akad. d. Wiss., math. naturw. Cl. 1885, vol. xcii, pp. 443–447.

8. THE INFLUENCE OF ATMOSPHERIC PRESSURE ON BLOOD-PRESSURE

The striking parallelism between the blood-pressure and the external pressure to which the body is subjected, has been exhaustively studied by Crile,[2] and his results are of the greatest interest in connection with the treatment of surgical shock. He has shown in many experiments a rise in pressure in practically all the arteries of the body when the surrounding pressure is increased, in diastolic pressure apparently more than systolic. The rise is proportional to the height of the external pressure, and occurs equally after division of the cervical cord. It is therefore a direct mechanical effect, and not due to reflex vaso-constriction. It is least in the arteries within the bony cavities of cranium and thorax. The height at which the pressure will be maintained depends only on the reserve force of the heart. He observed a corresponding fall on diminishing atmospheric pressure. This effect of rarefied air was more thoroughly studied by Lazarus and Schirmunski,[3] who found, not only a uniform sinking of carotid pressure with the atmospheric pressure, but a continuance of the low pressure for a time after normal conditions had been restored. Only when pronounced muscular twitching occurred did they miss the fall.

---

[1] A diagram illustrating the effect of asphyxia on the various nerve centres may be found in Tigerstedt's Lehrbuch der Physiologie des Menschen, second edition, 1902, vol. ii, p. 298.

[2] Crile, George W. Blood-pressure in Surgery. Philadelphia, 1903, pp. 283–286.

[3] Lazarus und Schirmunski. Ueber die Wirkung des Aufenthalts in Verdünnter Luft auf den Blutdruck. Zeitschr. f. klin. Med., 1884, vol. vii, p. 299.

Crile's experiments on the effects of breathing air at different pressures were inconclusive because too great and sudden changes were produced. Bartlett[1] has investigated the effects of breathing rarefied air in rabbits. He finds that the aortic pressure falls with that of the inspired air, after a sufficient negative pressure has been reached. The point at which the drop begins varies, in different individuals, between 10 and 15 mm., and is marked at a pressure of −15 to −25 mm. At these lower figures the pulmonary circulation is slowed and stagnation in the capillaries, with lung swelling, occurs.

9. THE INFLUENCE OF MUSCULAR WORK ON BLOOD-PRESSURE

The conditions of direct manometric measurement are not compatible with much exertion on the part of the animal, and little experimental evidence on the subject exists. Marey[2] taught that work lowered pressure, quoting an experiment on a horse, in which a run of ten minutes reduced the mean carotid pressure from 108 mm. to 102 mm., an insignificant difference. For such a fall to occur, vascular dilatation would have to be more marked than the increase of cardiac energy.

Tangl and Zunst[3] have succeeded in obtaining trustworthy results in a few dogs. These were made to run up steps, and the maximum and minimum carotid pressures measured at intervals. From these the absolute mean pressure was calculated. They found a uniform rise in mean pressure of 6 to 23 mm. Very fast running caused an extreme elevation, in two instances from 115 mm. to 235 mm., and from 120 mm. to 242 mm., respectively. There seems no reason to question their general results, which prove the usual tendency of muscular work to raise pressure.

---

[1] Bartlett, Frederic H. On the Variations of Blood-pressure during the Breathing of Rarefied Air. Am. Jour. of Physiol., 1903, vol. x, p. 143.

[2] Marey, E. J. La circulation du sang à l'état physiologique et dans les maladies. Paris, 1881, p. 343.

[3] Tangl, F., und Zunst, N. Ueber die Einwirkung der Muskelarbeit auf den Blutdruck. Arch. f. d. gesam. Physiol., 1898, vol. lxx, p. 544.

PART II.—TECHNICAL

CHAPTER III

THE INDIRECT MEASUREMENT OF BLOOD-PRESSURE

1. **Introduction.**
    - A. The palpation of the pulse. Methods of judging tension and their defects.
    - B. Sphygmographic methods of estimating blood-pressure.
    - C. Other methods which apply pressure through a solid block.
2. **The development of sphygmomanometers applying pressure to the artery through a fluid medium.**
    - A. The instrument of v. Basch, determining the pressure necessary to obliterate the pulse (systolic pressure), and Potain's modification.
        - a. Errors of the v. Basch and Potain sphygmomanometers.
    - B. The origin of sphygmomanometers registering maximum pulsation (diastolic pressure).
        - a. Marey's first instrument. Hürthle's elaboration.
        - b. Marey's second instrument.
        - c. Mosso's apparatus.
        - d. Oliver's hæmodynamometer.
        - e. Hill and Barnard's pocket sphygmometer.
    - C. Change in color of the skin as a means of determining systolic pressure.
3. **The method of circular compression by air** (Riva-Rocci. Hill).

4. Experimental verification of the method of circular compression.

A. Influence of the inner wall of the tube.

- a. Influence of faulty adjustment.

B. Influence of the tissues.

- a. Relation of width of armlet to circumference of limb. (v. Recklinghausen.)
- b. Influence of the site of application.
- c. Influence of the vessel wall.
- d. Influence of muscular contraction.
- e. Influence of œdema.

C. Conclusions.

5. Validity of the several criteria.

A. Of systolic pressure.

- a. The return of the pulse wave under compression.
- b. The return of color to the skin of the blanched finger.
- c. Comparison of systolic pressures in brachial and digital arteries.

B. Of diastolic pressure.

- a. Maximum pulsation of the arterial wall.

C. Other criteria, not in clinical use.

CHAPTER III

THE INDIRECT MEASUREMENT OF BLOOD-PRESSURE

1. INTRODUCTION

THE impetus which Ludwig's invention of the kymographion gave to the study of circulatory physiology soon made itself felt in the field of clinical medicine. The introduction of the sphygmograph, which could be applied to the living patient, enhanced this interest, and the investigation of the finer characters of the pulse seemed about to yield valuable practical results. How little it has fulfilled that early hope we know. With the sphygmograph, however, came the first attempt to measure human blood-pressure, made by K. Vierordt in 1855. The distinction between the hard and the soft pulse had been made before, but from that day the terms acquired a new significance, and the clinic set itself the task of finding some means for assigning them a quantitative value, comparable with the results of the physiological laboratory. The history of the successive methods is a long one, not always free from rancor and jealousy, when a cherished instrument has done its best service in the unexpected way of suggesting a more perfect one; but more and more objective determinations have followed one another, until to-day the intelligent physician can no more afford to dispense with some form of sphygmomanometer, in the study of certain types of disease, than he could discard the thermometer and trust his trained sense of touch, to detect the variations of body temperature in a case of typhoid fever. That sphygmomanometry has the wide range of applicability or the importance of clinical thermometry, not even its most enthusiastic advocate would claim; but that we now possess several instruments, of sufficient accuracy and such ease of application, as to make their routine use a necessity for the educated and conscientious practitioner, seems to me proven.

A. The Palpation of the Pulse.—Methods of Judging Pressure and their Defects.—The main ground for such a belief lies in the extreme fallaciousness of that time-honored method, the palpation of the pulse, as a means of estimating the blood-pressure. How erroneous our former judgments were in this respect, the use of a sphygmomanometer for a few days will convince any one. The reason for it is simple and the cause is, from the nature of the method, unavoidable. Two ways of estimating the tension of the pulse are in vogue. The first consists in compressing the artery with the finger, until the pulse can be no longer perceived by a second finger, placed on the artery just below. From the considerations on page 10, it is plain that this is an attempt to test the systolic end pressure. The other procedure is, to increase gradually the pressure of the finger, or better fingers, upon the artery, noting the change in the pulse-wave all the while. In pulses of high tension, the size of the wave will be felt to increase with increasing pressure up to a certain point, diminishing only when considerable force is used; whereas, with normal or low tension, the amplitude of the pulse is reduced with very little application of pressure. In this way a knowledge of the diastolic lateral pressure is obtained, for reasons which will be discussed fully in connection with the instruments which measure this. In many patients, the larger variations in arterial pressure may be readily detected by either method, the latter being somewhat preferable, because less affected by the amount of tissue overlying the artery. Both fail, however, in a crucial point. Our muscular sense, upon which depends our judgment of the amount of pressure used, takes cognizance only of the total amount of force expended, not at all of such a mathematical ratio as amount of force to the unit of surface. Were arteries, or even radial arteries, all of equal calibre, the two quantities would be identical. Unfortunately there are not only wide variations in size in different individuals, but, in one and the same person, the radial or temporal artery changes in diameter markedly under varying local conditions. Since the total force exerted is proportional to the extent of surface of the artery, as well as to the pressure per unit surface within it (by Pascal's law), the larger the artery, the higher we shall think the pressure. This will be readily appreciated by any one, who will attempt to compare the apparent pressures in the radial and in the abdominal aorta, which, in reality, are nearly equal. It is also evident that our failure to detect increased pressure by the finger will be most unavoidable in the small, so-called wiry pulse, which often connotes an extreme grade of high tension.

B. Sphygmographic Methods of Estimating Blood-pressure.—Following Vierordt's[1] lead, more elaborate adjustments of weights in connection with Marey's sphygmograph were made by Förster, Landois, and Behier. Even as recently as 1897, Philadelphien devised his so-called "sphygmométrographe," and Levaschoff,[2] in 1901, employed a somewhat similar method. These instruments are of purely historic interest, for the sphygmograph is an instrument whose results are notoriously subjective, and dependent upon the observer who applies it. Apart from this, the pressure, in all of them, was applied to the artery through a solid block, and therefore they measure total pressure, as does the finger; not pressure per unit of surface. They served, however, to establish one criterion, as a measure of blood-pressure; viz., the pressure at which the pulse-wave is obliterated. This, we have seen, gives the systolic end pressure, since it is equivalent to attaching a manometer to the end of the artery at the point of compression. (See page 11.)

C. Other Methods which Apply Pressure through a Solid Block.—Waldenburg in 1877, and later Bloch, Verdin, Cheron, and Hoorweg,[3] brought forward different forms of apparatus, which all possess the same fatal defect of applying the pressure to the artery through a solid medium. Within a few years v. Frey has added to this list another, which requires twenty minutes for a single observation.[4]

---

[1] All methods described in this chapter, for which no references are given, may be found in the elaborate historical articles of N. Vaschide and J. M. Lahy, La technique de la mesure de la pression sanguine. Arch. gén. de Médecine, 1902, vol. ii, pp. 349–383, 480–501 and 602–639.

[2] Levaschoff, I. N. Vratch, St. Petersburg, 1901, vol. xxii, pp. 1433 and 1471.

[3] Hoorweg, J. L. Ueber die Blutbewegung in den menschlichen Arterien. Arch. f. d. gesam. Physiol., 1889, xlvi, p. 166.

[4] Hayaski, T. Vergleichende Blutdruckmessungen an Gesunden und Kranken mit den Apparaten von Gärtner, Riva-Rocci und Frey. Inaug. Dissert., Erlangen, 1901, Dec.

2. THE DEVELOPMENT OF SPHYGMOMANOMETERS APPLYING PRESSURE TO THE ARTERY THROUGH A FLUID MEDIUM

A. The Instrument of v. Basch, Determining the Pressure Necessary to Obliterate the Pulse (Systolic Pressure), and Potain's Modification.—The first man to perfect a sphygmomanometer suitable for clinical use, and free from gross inaccuracy, was Prof. v. Basch of Vienna. In 1876 he constructed his apparatus, which in its original form is shown in Fig. 7. In two fundamental respects it differed from all that had preceded it. One was the employment of a bag containing fluid as the compressing mechanism, thus obtaining pressure per unit of surface for the first time; the other was the use of a mercury manometer for the measurement of the pressure within the bag necessary to wholly compress the artery. In this way he made possible a comparison of results with the direct estimations of the physiologist.

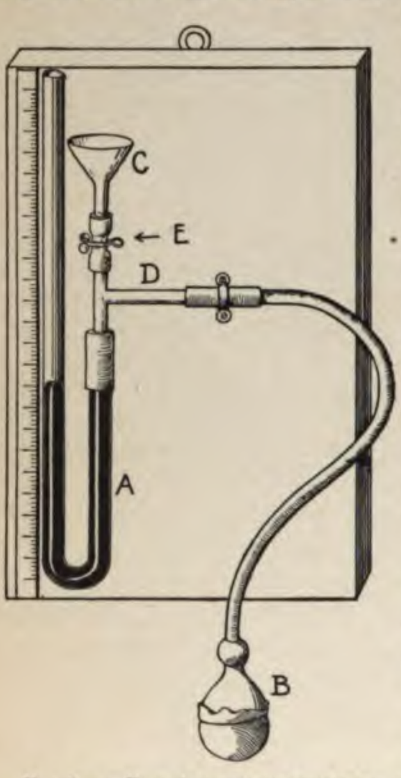


FIG. 7.—V. BASCH'S SPHYGMOMANOMETER. First model.

The essential parts of the v. Basch sphygmomanometer are seen in the cut. B is the so-called pelotte, originally a glass funnel covered with an elastic membrane, only slightly stretched. The pelotte is connected with the manometer (A) by non-distensible tubing and a T-canula (D). The vertical branch leads to a funnel (C), through which the pelotte and tubing are filled with water. At E is a pinch-cock, by which this communication can be cut off, after the mercury has come to equilibrium and the zero point of the manometer has been determined. The mode of application is as follows: An artery, temporal or radial, is selected, which lies closely upon bone, and pressure is made over it with the pelotte, care being taken that the compression shall be directly against the bone. As the pressure is increased, water is forced out of the pelotte into the closed arm of the manometer, which at each moment registers the exact pressure exerted over the artery. The pulse is felt with a finger of the opposite hand, just beyond the point of compression, or made visible by a lever held over the artery by a rubber bracelet. Just at the moment when it disappears, or, as most prefer, when it first returns as the pressure is lowered, the manometer is read off. This reading gives the pressure in millimeters of mercury necessary to obliterate the artery; that is, the systolic end pressure, plus certain other factors, which will be considered shortly.

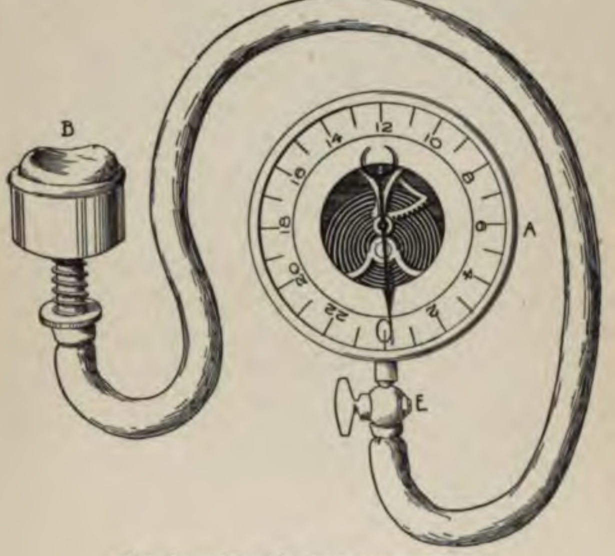


FIG. 8.—V. BASCH'S SPHYGMOMANOMETER.
Recent model.

The original instrument of v. Basch has undergone numerous modifications at his own hands and others. The most important were his introduction of a portable metal manometer, and the change in the pelotte, as shown in Fig. 8; and, in 1889, Potain's replacement of the water by air, the pressure of which is raised by a bulb connected with the circuit through an extra branch tube. Potain's sphygmomanometer, as shown in Fig. 9, was the basis of his extended clinical researches into arterial pressure,[1] and has a wide vogue in France; while the v. Basch instrument is still stoutly defended against all new-comers by its inventor and his pupils of the Vienna school. To them belongs the great honor of the pioneer work in the field of clinical sphygmomanometry, and to their efforts we owe the recognition of its value; but the lack of earlier acceptance by physicians in general was due to distrust of the absolute value of the results obtained with the instrument, and to the skill required for its use. v. Basch will always be remembered as the inventor of clinical sphygmomanometry, but his method must give place to its simpler and more accurate offspring.

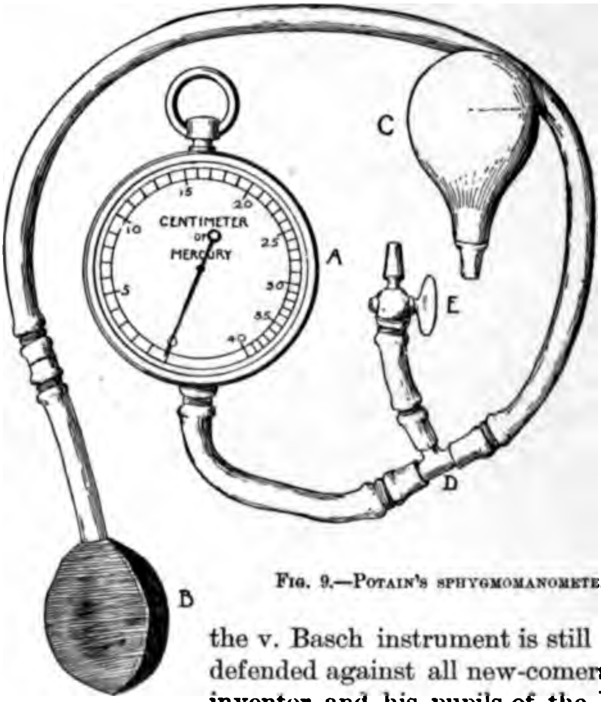


FIG. 9.—POTAIN'S SPHYGMOMANOMETER.

---

[1] Potain. La pression artérielle de l'homme à l'état normal et pathologique, Paris, Masson, 1902.

A. Errors of the v. Basch and Potain Sphygmomanometers.—These are due, not to defects in the instrument itself, but to the method of its application. It measures the pressure necessary to obliterate the lumen of the artery. Were the artery exposed, with no overlying tissue, and placed directly against the bone, this would equal the systolic end pressure plus the pressure required to close the empty artery. This, according to v. Basch, for the normal radial scarcely amounts to 1 mm. and even for sclerotic arteries is not much above 5 mm.[1] There may also be, v. Basch admits, a difference of 5 mm. between the point of disappearance of the pulse-wave and its return. In the living patient, however, the artery is covered by skin and a varying amount of adipose, which the author considers require 6 to 8 mm. additional pressure. These causes of overestimation, since they are small and more or less constant, and also because they must be present in any clinical method which gives systolic end pressure, could not be considered serious.

The damaging evidence, which v. Basch himself gives, relates to the effect of the varying position of the artery with reference to the bone. Only rarely did he find the pressure in temporal and radial nearly alike, in most cases the radial requiring 20 mm., and, in exceptionally badly placed ones, 60 mm. more for its compression. These readings were made by practised observers, and in unskilled hands would make a far worse showing. Potain, in his book (p. 36), cites several cases as showing the great difference in pressure which may exist in different arteries at the same moment. One, a young girl, gave 5.5 cm. for the temporal, 16.5 cm. for the radial; an adult, 12.5 cm., temporal; 19.5 cm., radial; and 17 cm., dorsalis pedis. He observed differences of 0.5 to 2 cm. between the two radial arteries. These figures contradict our established physiological knowledge concerning the pressure in different arteries (see page 30), and are unquestionably due to errors inherent in the method and to variations in the position of the artery, so that the pressure is not always applied directly over it and against the bone.

---

[1] v. Basch, S. Der Sphygmomanometer und seine Verwerthung in der Praxis. Berl. klin. Wochenschr., 1887, vol. xxiv, p. 181.

Tigerstedt[1] sums up these sources of error from v. Basch's figures as follows:

Lowest estimate; $1+5+6+20$ mm. $=32$ mm. overestimation.
In unfavorable cases; $5+5+8+60$ mm. $=78$ mm. "

He concludes, and rightly, that the absolute values found with such instruments can have no significance, but that they are suited to the estimation of the pressure changes which occur in the same individual, within reasonable periods of time.

I have gone thus at length into this subject, because one must exercise a similar criticism in the results of all other methods of indirect blood-pressure determination, in each of which some of the same sources of overestimation are operative.

B. The Origin of Sphygmomanometers Registering Maximum Pulsation (Diastolic Pressure).—a. Marey's First Instrument.—Hürthle's Elaboration.—At the same time that v. Basch brought out his instrument, Marey carried on experiments in his laboratory, from which our clinical methods of determining diastolic pressure have been developed. From them, besides, have come the plethysmograph and its congeners.

Marey's observations were upon the effect of compressing the blood-vessels from all sides, instead of from a single surface. His first apparatus was a tight metal box, filled with water, and large enough to hold the fore-arm and hand inserted through a rubber collar, which entirely closed the opening. The box was connected with a mercury manometer and a recording tambour, as well as with a reservoir, by means of which the pressure could be gradually raised. A glass window afforded a view of the enclosed hand. Marey noted that, as the pressure within the box was raised, the pulse waves became transmitted to the fluid and thus to the tambour. These pulsations increased in amplitude up to a certain point, then gradually declined again. Long before they had entirely ceased, however, the skin of the hand was seen to blanch, thus showing that the vessels had been collapsed and their systolic pressure overcome. Marey reasoned that, at the moment when the pulsations reached their maximum, the external pressure must be just equal to that within the blood-vessels;

---

[1] Tigerstedt. Lehrbuch d. Physiol. des Kreislaufes, Leipzig, 1893, p. 331.

their walls, being thus relieved of any tension on either side, would execute their maximum oscillations. He proposed this as a new criterion for the indirect measurement of blood-pressure; the external pressure at which maximum pulsation of the arterial walls occurs.[1] Subsequent investigation has shown that this corresponds with considerable accuracy to the diastolic pressure. This first instrument of Marey was much elaborated by Hürthle at a later date. As his apparatus is very cumbersome and suited only to laboratory use, it need not detain us here.

b. Marey's Second Instrument.—Marey, however, devised a second, smaller form (Fig. 10), which received a single finger and transmitted the pulsations direct to the mercury column; but the volume changes in one finger proved too slight to give satisfactory readings. It was discarded until, in 1895, Mosso, using it as a basis, constructed his admirable laboratory sphygmomanometer.

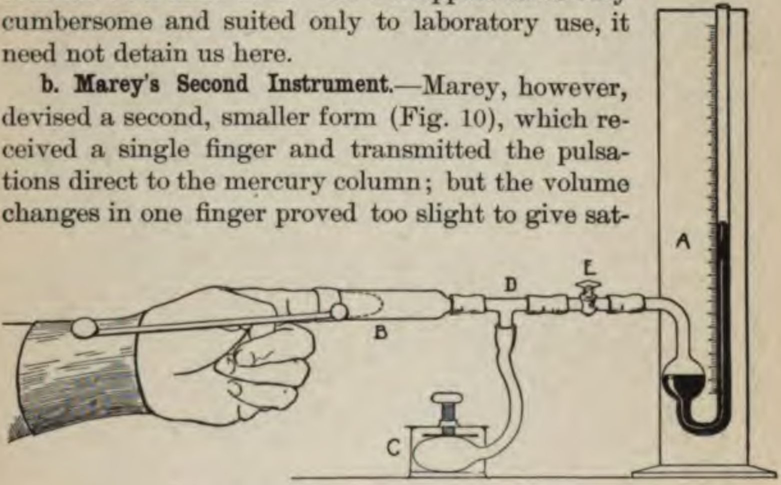


FIG. 10.—MAREY'S SECOND SPHYGMOMANOMETER.

c. Mosso's Apparatus.[2]—The essential parts of this rather complicated apparatus, shown in Fig. 11, are: 1st, Four metal tubes (BB) enclosing loose rubber glove fingers, into which are thrust the two middle fingers of each hand. 2d, A recording mercurial manometer (A) connecting with the tubes (BB) through the tube (D), filled with water. 3d, A pump (C), whose piston is slowly depressed by turning a crank. The whole apparatus is filled with water from the bottle (F), the air driven out through the cock (E) and the pressure raised, either gradually or by steps of 10 mm. each, until the tracing of the manometer attains its maximum height. This instrument is only suited to laboratory use, and shares, with others which determine pressure in the small vessels of the finger, certain disadvantages to be considered later. It has yielded much accurate information, however, and suggested the evolution of our present clinical sphygmomanometers which measure diastolic pressure.

---

[1] For tracings illustrating this see Figs. 18 and 20.

[2] Mosso, A. Sphygmomanomètre pour mesurer la pression du sang chez l'homme. Arch. Ital. de Biol., 1895, vol. xxiii, p. 177.

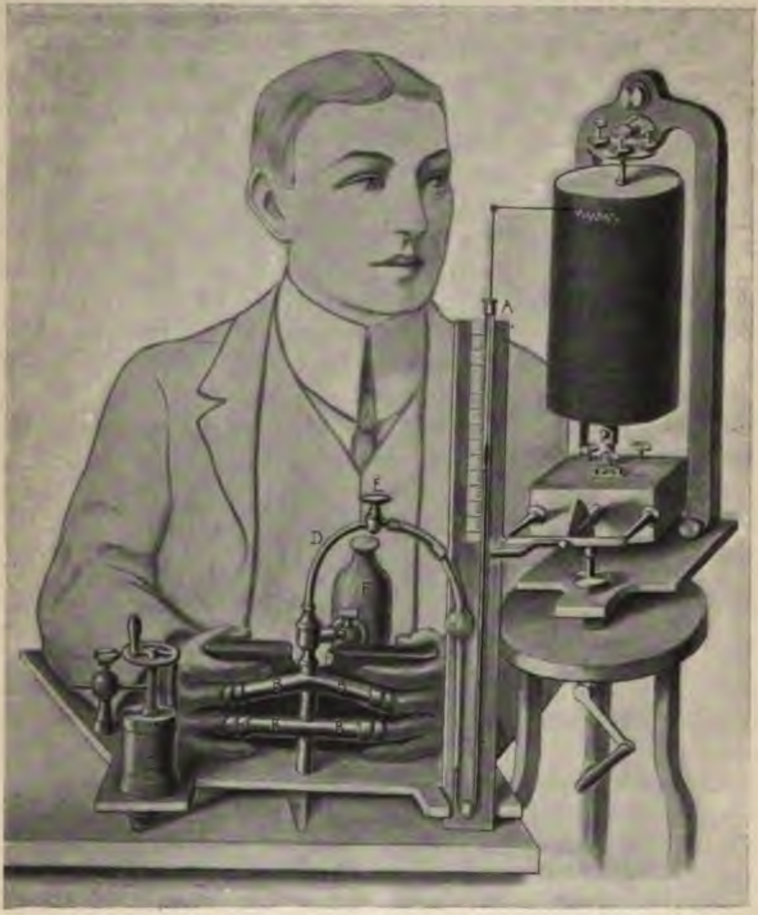


Fig. 11.—Mosso's sphygmomanometer.

d. Oliver's Hæmodynamometer.[1]—Oliver, in 1898, brought forward a simple instrument for the measurement of arterial pressure, consisting of a circular spring manometer, graduated in mm., Hg., with a hollow metal stem terminating at its foot in a small rubber bag filled with fluid. With this, compression is made over any exposed artery until the oscillation of the needle on the dial attains its maximum, which Oliver thought corresponded to mean arterial pressure. The instrument marks a return to the v. Basch method of compression, and therefore is liable to all the errors of faulty application which we have considered; besides, its manometer is not trustworthy unless standardized frequently. It cannot, therefore, be ranked with the modern instruments, though its construction is simple and ingenious.

e. Hill and Barnard's Pocket Sphygmometer.[2]—This little instrument may fit a waist-coat pocket, but has scarcely more value than the finger as a measure of pulse tension. It consists of a vertical glass tube six inches long, ending above in a small bulb with glass stop-cock. Below it expands into an open cup, which is covered by a rubber membrane, like v. Basch's pelotte. The tube is filled with glycerine colored by chromic acid, and is graduated empirically in mm. Hg. To use it, the stop-cock is closed and the tube, held vertically, is pressed down on the artery until the pulsation becomes maximal.

C. Change in Color of the Skin as a Means of Determining Systolic Pressure.—Indirectly, Marey's observations suggested another criterion for the determination of systolic pressure; viz. the change in color of the skin. This was used at about the same period, 1875, by v. Kries, in measurements of capillary pressure, but has recently come into prominence through a popular sphygmomanometer which embodies it as its principle, the tonometer of Gärtner.

---

[1] Oliver, George. A Simple Pulse Pressure-Gauge. Jour. of Physiol., 1897–98, vol. xxii, p. 51. A Contribution to the Study of the Blood and Blood-pressure, Geo. K. Lewis, London, 1901, pp. 104 to 272.

[2] Hill, L., and Barnard, H. A Simple Pocket Sphygmometer for Estimating Arterial Pressure in Man. Jour. of Physiol., 1898, vol. xxiii. Proceed. of Physiol. Soc., p. iv.

3. THE METHOD OF CIRCULAR COMPRESSION BY AIR (Riva-Rocci. Hill)

None of the sphygmomanometers introduced prior to 1896 complied with all the essential requirements for general use by physicians; reasonable accuracy, freedom from subjective errors, ease of application, and portability. The more exact instruments, such as Mosso's, were very cumbersome; the light and compact ones had all the errors of the v. Basch apparatus, and frequently more. This technical difficulty was first overcome by Riva-Rocci,[1] and by Hill,[2] the former in an instrument for the measurement of systolic pressure, the latter for diastolic. Though the former published first, it is questionable whether the credit of the new device does not belong to Hill.

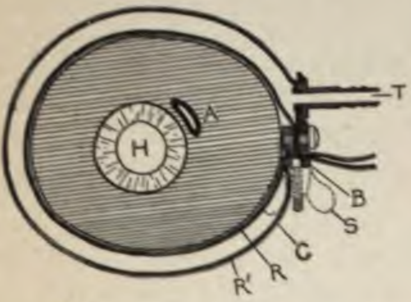


Fig. 12.—Cross-section of arm, Riva-Rocci armlet.

R = inner wall of rubber tube.
R′ = outer wall of rubber tube.
C = silk cover.
B = clamp, fastened by
S = screw.

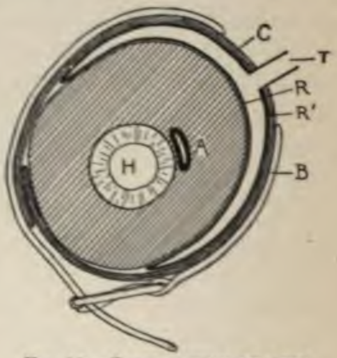


Fig. 13.—Cross-section of arm, Hill and Barnard armlet.

R = inner wall of rubber bag.
R′ = outer wall of rubber bag.
C = leather cuff.
B = strap, fastened by buckle.

In each T = tube leading to manometer.
A = brachial artery.
H = humerus.

The important feature in both instruments was the adoption of a rubber tube or bag, encircling the arm and inflated by means of a bulb or pump, as the compressing mechanism (see Chapter IV). We have seen that the most serious error of the v. Basch sphygmomanometer was due to the difficulty of compressing the radial artery directly against the bone with the small and awkward pelotte.

---

[1] Riva-Rocci. Un nuovo sfigmomanometro. Gazz. Med. di Torino, 1896, No. 50, 51.

[2] Hill, Leonard, and Barnard, Harold. A Simple and Compact Form of Sphygmometer or Arterial-pressure Gauge devised for Clinical Use. British Med. Jour., 1897, vol. ii, p. 904.

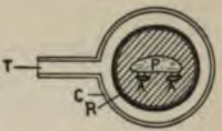


Fig. 14.—Cross-section of finger with Gartner ring.

R = inner rubber membrane.
C = metal ring.
T = tube leading to manometer.
AA = digital arteries.
P = phalangeal bone.

The accompanying cuts (Figs. 12 and 13) show the new method. The Riva-Rocci tube completely encircles the arm and fastens with a special clamp, while the armlet of Hill surrounds more than half of it and is held in place by an outer leather cuff and buckle. In either case the pressure is everywhere exerted at right angles to the surface of the arm, and therefore must compress the artery equally from three sides against the underlying bone.

Gärtner,[1] in his tonometer, uses a practically identical method of compressing the finger.

4. EXPERIMENTAL VERIFICATION OF THE METHOD OF CIRCULAR COMPRESSION

If the results of the various sphygmomanometers using the method of circular compression are to be accepted as having an absolute, or nearly absolute, value, the method must be found to comply with certain fundamental requirements.

A. Influence of the Inner Wall of the Tube.—The pressure exerted upon the limb must be exactly the pressure within the tube. This question was thoroughly investigated by Gumprecht,[2] who found a substantial correspondence between the internal pressure and the pressure exerted. There was no increasing loss as the pressure was raised, as would have been the case had the elasticity of the tube itself been called into play.

---

[1] Gärtner, G. Ueber einen neuen Blutdruckmesser (Tonometer). Wien. med. Wochenschr., 1899, vol. xlix, p. 1412.

[2] Gumprecht. Experimentelle und klinische Prüfung des Riva-Rocci'schen Sphygmomanometers. Zeitschr. f. klin. Med., 1900, vol. xxxix, p. 377.

a. Influence of Faulty Adjustment.—This only holds good when the armlet is properly applied, that is, snugly, but not so tightly as to exert any pressure before inflation. I have frequently found the reading as much as 20 mm. to 30 mm. too high, on account of loose adjustment of the cuff. This is especially prone to happen in those instruments in which the cuff is buckled on, one hole being too loose and the next too tight. It is more easily avoided with the original Riva-Rocci clamp, or the type of buckle adopted in my modification. With the compressing finger ring of the Gärtner tonometer, it becomes much more difficult to fit each case, without carrying a large number of rings of different sizes. Martin,[1] who has investigated the point carefully and gives convincing experimental evidence, thinks the error from erroneous application much greater than with the Riva-Rocci, and that the adaptation of ring to finger must be very exact, even to differences of 1 mm. in circumference. For this reason he has devised a small modified Riva-Rocci tube, of 2.5 cm. width, for the finger, and obtains accurate results with it. Wolf[2] found a similar influence of the calibre of the ring, smaller rings giving higher readings on an artificial rubber finger filled with mercury and connected with a manometer tube. Of seventy-two observations on the two hands, in only one half did the readings correspond.

B. Influence of the Tissues.—The pressure exerted by the tube upon the arm must either be transmitted without loss to the blood-column within the artery, or the loss must be a constant factor, capable of calculation. The earlier investigations on this point led to the conclusion that there was a certain loss, due to the compressibility of the tissues, which was called the elasticity error. It was variously estimated. Gumprecht placed it at 30 mm. for moderate pressures and rising to 40 or 50 mm. at high pressure; Hensen[3] at 10 to 15 mm. in adults, 3 mm. in children. The latter called attention to the important fact that, were the error at all considerable, it would be very noticeable in a long series of observations on arms of varying size. It must increase proportionally to the area of cross-section, not the circumference of the arm, therefore if there were an error of many mm. Hg. in normal arms, it should be impossible to find moderate pressures in large arms. This is not the case in my experience or that of other observers.

---

[1] Martin, Alfred. Technisches über das Riva-Rocci'sche Sphygmomanometer und Gärtner's Tonometer. Münch. med. Wochenschr., 1903, vol. l, pp. 1021 and 1072.

[2] Wolf, H. Experimentelle Untersuchungen über die Blutdruckmessungen mit dem Gärtner'schen Tonometer. Wien. med. Presse, 1902, vol. xliii, pp. 1349 and 1395.

[3] Hensen, H. Beiträge zur Physiologie und Pathologie des Blutdrucks. Deutsch. Arch. f. klin. Med., 1900, vol. xlvii, p. 437.

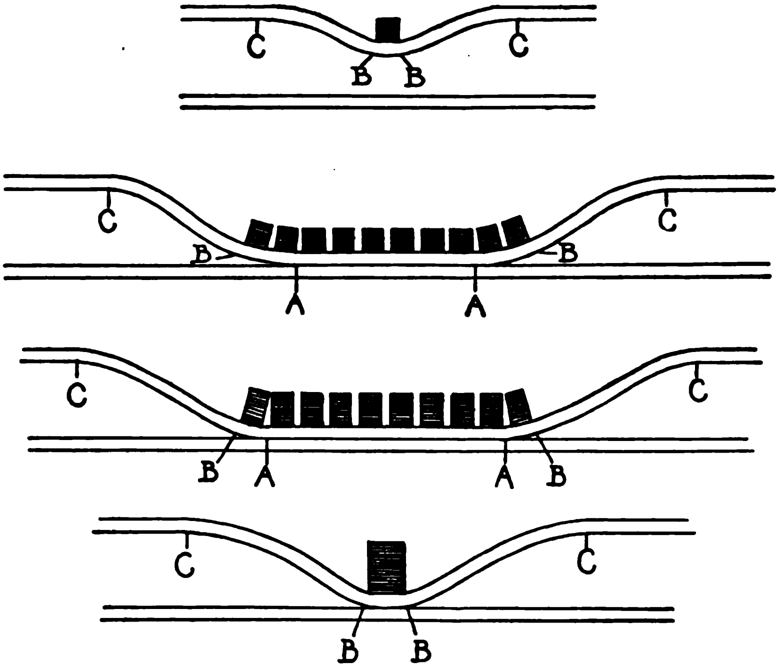


Fig. 15.—Distortion of a tube by blocks laid on it.
(From v. Recklinghausen, p. 87.)

a. Relation of Width of Armlet to Circumference of Limb. v. Recklinghausen.—v. Recklinghausen,[1] in his excellent critical studies, approached the question from another standpoint.

He believed that the soft parts exerted no influence whatever, and investigated the effect of the length of artery compressed. The foregoing diagram (Fig. 15) shows his method of reasoning. It is evident that the portions of tube B C carry some of the weight B. If a number of weights of the same size be laid along the tube, not increasing the pressure to the square inch, the outer blocks, on AB only, will feel this pull, while the central ones will exert their full pressure upon the portion of tube directly beneath them. With this in view he constructed armlets of different widths, and found that a certain proportion must be maintained between circumference of arm and length of it compressed. For ordinary arms of 24 cm. circumference, a 10 cm. cuff sufficed, and 12 to 15 cm. was adequate for almost any size. When too small an armlet was used, the readings were considerably higher than with the proper width. Since the Riva-Rocci arm-piece has a width of at most 5 cm. and, when distended, becomes circular and is in contact with an even smaller surface, v. Recklinghausen thought that he had discovered the cause of the so-called elasticity error of former observers and its cure. In this I believe he was fully justified. His results have been substantiated recently by Martin, who adopted a 10 cm. width, and Erlanger,[1] who has modified his very exact apparatus by the substitution of a 12 cm. cuff for his previous one. In experimenting with dogs, estimations from the thigh, with cuffs of 3.5 cm. width, showed errors ranging as high as 50 mm. Hg.; but with 9 cm. width the error, under normal conditions, was never greater than 10 mm. Whatever error was present entered equally into the determination of systolic pressure (obliteration of pulse), and diastolic pressure (maximum pulsation). In this country, besides Erlanger, Stanton[2] has introduced an armlet of 8 cm. width in his instrument, and the author one of 12 cm. in his new form. With it I have found readings, in high tension pulses, as much as 60 mm. lower than with the 5 cm. tube. This increase of the error at high pressures, which certainly occurs and which Gumprecht first pointed out, I believe is due to the fact that the compressing surface grows smaller the higher the pressure is raised, since the tube becomes more circular in cross-section as it distends.

---

[1] v. Recklinghausen, H. Ueber Blutdruckmessung beim Menschen. Arch. f. exper. Pathol. u. Pharmakol., 1901, vol. xlvi, p. 78.

---

[1] Erlanger, Joseph. A Study of the Errors involved in the Determination of the Blood-pressures in Man, together with a Demonstration of the Improvements in the Sphygmomanometer suggested thereby. Am. Jour. of Physiol., 1904, vol. x; Proceed. of Am. Physiol. Soc., p. xiv.

[2] Stanton, W. B. A Practical Clinical Method for determining Blood-pressure in Man, with a Discussion of the Methods Hitherto employed. Univ. of Penn. Med. Bull., 1903, vol. xv, p. 466.

v. Recklinghausen gives the following table, which illustrates the difference between the old size of compressing surface and that recommended by him.

SIMULTANEOUS DETERMINATIONS WITH THE 5 CM. WIDE CUFF OF RIVA-ROCCI ON ONE ARM AND A 12 CM. WIDE CUFF ON THE OTHER

Person H., sitting, directly after dinner. Pressures in mm. Hg. Maximum circumference of each arm 24½ cm. (Thicker arms would naturally give greater differences.)

	BROAD CUFF	RIVA-ROCCI'S CUFF
Pulse returns	Right arm 90– 96	Left arm 116
Pulse returns	Right arm 92–100	Left arm 114
Pulse disappears	Right arm 90– 98	Left arm 112
Pulse returns	Right arm 92– 98	Left arm 114
Pulse returns	Left arm 96–104	Right arm 118
Pulse returns	Left arm 94–104	Right arm 120
Pulse disappears	Left arm 104–108	Right arm 120
Average	94–101	116

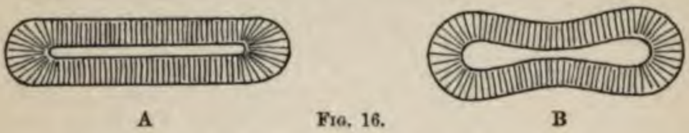
With the lower pressure values of the first column the mercury manometer showed marked pulsations, with the higher ones of the second column it stood quite still. (After v. Recklinghausen, loc. cit., p. 110.)

With the finger ring of the Gärtner tonometer he found a similar influence of the extent of compressing surface, and recommends a ring covering the whole proximal phalanx. Martin does not wholly agree in this, finding 2.5 cm. sufficient, instead of the 1.5 cm. width of the original Gärtner ring.

b. Influence of the Site of Application.—In all blood-pressure determinations, whether by direct or indirect methods, it is essential that the measurement be taken at the level of the heart. The reason for this is simple. If the observation be made at a point, let us say, 10 cm. lower than the heart, we shall measure not only the blood-pressure, but the weight of a column of blood 10 cm. high in addition. This equals about 8.5 mm. of mercury. If the point be above, a corresponding error will be introduced in the negative direction.

With the 12 cm. cuff it is, of course, immaterial on what segment of the limb it be placed, so long as it can be adapted smoothly to it. Determinations may be made equally on arm or thigh, or even on the calf. In children, whose arms are very small, the thigh is to be preferred. This obviously necessitates the recumbent posture to keep the level of the heart.

The Gärtner method of finger compression is not so fortunate in this respect. Martin found that the ring must be placed exactly on the middle phalanx, as Gärtner originally directed, under penalty of a possible error of 10 mm. or more. He explains the results of many of the critics of the tonometer, who have found marked variation in simultaneous readings from different fingers, as due to this technical error.


A FIG. 16. B

A. Manner of closure, normal artery.
B. Manner of closure, sclerotic artery, leaving open corners.

(After v. Recklinghausen, p. 104.)

c. Influence of the Vessel Wall.—Since the experiments of v. Basch already quoted (see page 49) showed that a pressure of 1 mm. was always sufficient to close the lumen of an excised radial artery, the error from this source may be absolutely neglected. That a sclerotic vessel may offer considerable resistance to compression is, however, a common belief, which I do not think is justified. v. Basch found the empty sclerotic radial could be compressed by 5 mm. Hg. The digital arteries should give an even lower figure, but that the larger brachial would offer more resistance seems plausible. Hensen,[1] from an observation on a dying consumptive with moderate arteriosclerosis of the brachial, in whom he found a blood-pressure of 30 to 40 mm., argued that, since there was still some internal pressure in the vessel, the error due to rigidity of its walls could not have exceeded 20 mm. I am inclined to doubt the accuracy of touch which can detect a pulse of such small volume as a systolic pressure of 10 to 20 mm. would imply, and, were the return of the pulse really palpable, should consider the overestimation to have been less than 10 mm. v. Recklinghausen[1] thinks it more probable that, with marked thickening of the artery, its walls do not collapse in the same manner as the normal ones, when the internal pressure has been equalized, but leave a small opening at each side through which a rudimentary pulse-wave might pass (see Fig. 16). In such cases he suggests leaving these rudimentary waves out of consideration and using the first well-developed pulse as the criterion. In this I am inclined to agree with him.

---

[1] Hensen. Loc. cit., p. 505.

The problem will be discussed again later, in considering the complicated relation of arterio-sclerosis to blood-pressure; but my personal belief, based on a number of cases of marked thickening or calcification of the large arteries, in which I have failed to find abnormally high systolic pressures, is, that errors from this source, with the wide armlet, and using the first fully formed pulse as a guide, have little significance.

d. Influence of Muscular Contraction.—In connection with this loss of pressure in transmission through the tissues, there are certain avoidable sources of error. Muscular relaxation on the part of the subject must be complete. Hensen[2] found the same pressure on the two sides in fresh hemiplegics with flaccidity; showing that normal muscle tonus during relaxation is without influence. Muscular contraction, however, at once introduces an obstacle to the compression of the artery, which the same observer proved might cause an overestimate of 5 mm. to 80 mm., depending upon the force used. This renders accurate observations impossible in spastic conditions, tetanus, marked subsultus, and all states of heightened muscular tone.

e. Influence of Œdema.—Œdema is a similar source of error, for some of the pressure is expended in squeezing the fluid out of the tissues, so that the tube gradually produces a groove in the limb. Hensen found the reading, in an arm with slight œdema, 20 mm. higher than in the opposite one. It is wiser not to attempt determinations when it is present. On the same ground, a change in the normal elasticity of the tissues, the experimental verifications of sphygmomanometers on the cadaver cannot be accepted.

---

[1] v. Recklinghausen. Loc. cit., p. 104.

[2] Hensen, H. Beiträge zur Physiologie und Pathologie des Blutdrucks. Deutsch. Arch. f. klin. Med., 1900, vol. xlvii, p. 437.

C. Conclusions.—As a result of the critical investigations we have considered, the essential accuracy of the method of circular compression has been demonstrated. The use of an armlet of 12 cm. width, adjusted, with reasonable care as to tightness, anywhere upon the arm or thigh, enables us to exert upon the main artery of the limb a pressure nearly or quite equal to that within the armlet. The maximum error by this method is 10 mm. Hg., due to loss in transmission through the tissues.

The method, as applied to the finger (Gärtner tonometer), requires more care in the accurate fitting of the ring to the middle phalanx, and seems to give less constant results, but is still of sufficient reliability for clinical purposes. Since the pressure within the ring, or armpiece, can be exactly measured by a manometer, the technical requirements for accurate clinical measurements of blood-pressure have been fulfilled. It remains only to prove that our external indicators of the equalization of certain definite pressures within the artery, systolic and diastolic, correspond with the event.

5. THE VALIDITY OF THE SEVERAL CRITERIA

A. Of Systolic Pressure.—a. The Return of the Pulse-Wave Under Compression.—This, we have seen, was the original criterion of Vierordt and v. Basch. Formerly it was questioned whether the moment of disappearance or of reappearance should be used. There is no doubt, however, that the moment of return is more sharply defined and more easily detected, and it is now the generally accepted standard.

If, during the gradual release of the pressure on the arm, tracings be taken from the radial artery with a sphygmograph, as in my earlier work on this subject[1] and Masing's,[2] or, like v. Recklinghausen,[3] a volume trace made from the forearm, the first few waves to reappear will be barely visible; then, after only a few mm. fall in the pressure, larger pulse-waves will suddenly show, as is readily seen in Fig. 17. The last writer calls the small waves rudimentary, the later large ones, well-developed. The detection of the rudimentary waves requires skill and care, as well as a favorably placed artery. Hensen thinks it easier to detect them at the bend of the elbow, but I have found this an awkward procedure, and even more unsatisfactory in stout arms than palpation at the wrist. Evidently, therefore, in some cases the well-developed waves are the first felt, and with the novice this is the rule. The underestimation I do not believe can exceed 5 mm., where due care is used, except with irregular or very rapid pulses, since the points of disappearance and return may usually be determined within 5 mm. of one another. Cook and Briggs,[1] who take the mean

---

[1] Janeway, Theodore C. Some Observations on the Estimation of Blood-pressure in Man, with Especial Reference to the Results obtained with the Newer Sphygmomanometers. N. Y. Univ. Bull. of the Med. Sci., 1901, vol. i, p. 105.

[2] Masing, Ernst. Ueber das Verhalten des Blutdrucks des jungen und des bejahrten Menschen bei Muskelarbeit. Deutsch. Arch. f. klin. Med., 1902, vol. lxxiv, p. 253.

[3] Loc. cit., p. 103.

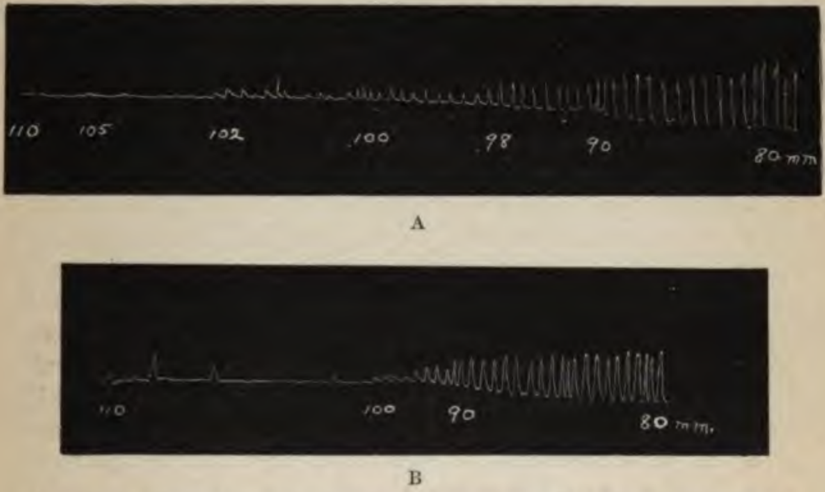


FIG. 17.—TRACINGS FROM THE FOREARM BY ERLANGER'S SPHYGMOMANOMETER DURING THE RELEASE OF PRESSURE IN ANOTHER INSTRUMENT ON THE UPPER ARM.

A. Slow release. Rudimentary waves at 102 mm., well-developed at 98 mm. The pulse, at 102 mm., was palpable, with care.

B. Fast release. Rudimentary waves at 100 mm., well-developed at about 90 mm. A few abnormally large pulses came through above 100 mm. The pulse could scarcely have been felt above 95 mm.

---

[1] Cook, H. W., and Briggs, J. B. Clinical Observations on Blood-pressure. Johns Hopkins Hosp. Rep., 1903, vol. xi, p. 451.

of these two points, do not consider readings satisfactory that differ by more than 2 or 3 mm.

One technical point must be insisted on. The pressure must be lowered very gradually, for, not only the rudimentary pulses, but several subsequent ones will be missed, if the mercury be allowed to fall 5 or 10 mm. at a time. (See Fig. 17.) It is always wise, so soon as the return of the pulse is perceived, quickly to raise the pressure the few mm. Hg. necessary to again obliterate it and make a second determination, taking the average of the two. For this purpose the instruments in which pressure may be rapidly changed in either direction with one hand (v. Recklinghausen, Erlanger, Stanton, Janeway) have a great advantage.

To obviate the subjective element in the determination, Erlanger has proposed a method of rendering the return of the pulse visible, which he calls "letting the cuff feel the return of the pulsation." Having raised the pressure in the sphygmomanometer considerably higher than maximum arterial pressure, he brings the recording lever of the tambour, in his instrument, to bear on the drum. The lever is depressed and shows a small pulse. The pressure is then allowed to fall gradually and the pulsations increase slightly, while the lever rises. At the instant the pressure in the armlet falls below the systolic pressure in the artery, the pulsation shows an abrupt increase in amplitude (see Fig. 20). This method, he has satisfied himself, is both accurate and objective. He claims that the readings so obtained are 5–15 mm. higher than when the finger is used to detect the return of the pulse. This may hold good when the fully-developed pulse is considered, but, with slow release of pressure and a little skill, the rudimentary waves may be felt, and I have never found my readings more than 2–5 mm. below simultaneous ones by his method. An objection to his method, for routine clinical use, is that it consumes more time, and requires quite as much skill. In pathological pulses, the change may be very indistinct. It is, of course, only applicable with a graphic instrument.

Apart from the adequacy of the sense of touch to detect these slighter waves, however, we must question whether the pulse passes through the compressed portion of the vessel the instant the pressure without falls below that within the artery.

With the broad armlets there must be 8 or 10 cm. of artery collapsed, and the first pulse-waves might lose their small initial surplus of pressure in overcoming friction, before reaching the wrist or even the elbow. This v. Recklinghausen showed was undoubtedly the case. By his very ingenious method of "Staircase curves," he was able to measure the absolute systolic pressure, and, in one curve which he figures, the first developed pulse he could detect came through at a pressure 6 mm. Hg. (8 cm. water) lower. This difference he calls the extra pressure required to open the path. Some consider the existence of this cause of low measurements good reason for using the average of the pressures found at the moment of disappearance and of reappearance of the pulse, the former being invariably higher; nevertheless, the disappearance is so much more gradual and misleading than the return, that most observers agree in preferring the definite end reaction with a small, fairly constant error. It may further be urged, that the probable underestimation of 5 mm. + 6 mm. = 11 mm. in the criterion used, may serve to cancel the equally probable overestimate of 10 mm., due to the resistance of the tissues and the arterial wall. This reasoning is certainly not sound where absolute values are essential, yet the variations in blood-pressure from slight causes during the course of an observation are of such extent, that this error of observation is immaterial. A change of 5 mm. or even 10 mm. in blood-pressure, unless all the conditions are absolutely safeguarded, affords no basis for deduction, and could only be allowed weight where the observations followed one another very closely. In that case the error would be absolutely constant and therefore negligible.

In connection with the measurement of the pressure variation of the pulse-wave, where this criterion is used for systolic pressure and the principle of maximum oscillation for diastolic, it may be urged that the latter measures lateral arterial pressure, while the method we are considering gives end pressure (see page 45). This is true, but of no practical importance. The figures of Tigerstedt[1] show that not more than $\frac{1}{100}$ of the work of the heart is expended in imparting to the blood-stream its velocity, the entire remainder being required to overcome aortic pressure. Hence, at a pressure of 160 mm. Hg., the difference between lateral and end pressure would only amount to 1 mm.

---

[1] Tigerstedt, Lehrbuch, p. 153.

A final point remains to be mentioned, namely, the well-recognized effect of compression of a limb in raising blood-pressure. This rise is general, since it is found as well in the opposite arm, and is unquestionably due to a central vaso-motor reflex, increasing peripheral resistance. It may appear within one minute as a rise of perhaps 5 mm., and, if compression is long maintained, can cause a rise of 20 mm. Hg. in exceptional cases. It must be carefully avoided, by working quickly and allowing an interval to elapse between every few determinations. It will be more troublesome in the estimation of diastolic than of systolic pressure, since the latter may be easily determined within half a minute.

In conclusion, one great advantage of this criterion must be considered. Measuring with fair exactness the systolic end pressure in the brachial artery, it gives us the systolic lateral pressure within the subclavian, since brachial and axillary are continuous in direction, and therefore a near approximation to systolic lateral pressure in the aorta. This, combined with estimation of diastolic lateral pressure in the brachial, which is practically the same as aortic diastolic pressure (see page 31), gives the best insight into actual variations of systemic blood-pressure.

b. The Return of Color to the Skin of the Blanched Finger.—This very ingenious method of making the moment of return of the pulse visible, though derived originally from Marey's observations, in its practical application we owe to Gärtner. As yet it is used by his instrument alone. His original statement was, that one obtained in this way the mean blood-pressure, basing his belief on comparison of readings thus made in a dog's tail, with direct measurements of its mean carotid pressure.[1] Such correspondence was, of course, purely accidental, since the method is theoretically identical with the detection of the return of the pulse after compression of the arm. The pneumatic ring is first placed on the middle phalanx, the finger is then rendered bloodless and the pressure in the manometer and ring raised to a point above systolic blood-pressure. It is then allowed to fall gradually until the white finger-end suddenly flushes. In many cases, if the pressure be released sufficiently slowly, a deeper flush appears a few mm. Hg. lower. With the flush, a throb, synchronous with the heart-beat, is felt by the patient. This second flush corresponds to the first well-developed pulse of the Riva-Rocci method.

---

[1] Gärtner, G. Wien. med. Wochenschr., 1899, vol. xlix, p. 1412.

Gärtner claimed for this criterion that it gave the subjective part in blood-pressure determinations to our most accurate sense, sight, while the Riva-Rocci method depended upon our rather fallible touch perceptions. The truth of this is unquestioned, except under certain conditions. By artificial light, and in the skin of the negro, or the very anæmic, color judgments are very difficult. Besides, many observers[1] agree that the flush frequently occurs so gradually as to be valueless as a guide. Hayaski takes the subjective throb as his index; in other words, he allows the patient to feel the return of his own pulse, and Gärtner himself speaks of the usefulness of the procedure. This is even more subjective than the palpation of the pulse by the observer, and requires co-operation on the part of the patient only possible in the intelligent and completely conscious. Martin finds the pressure corresponding to the throb in the highest degree dependent on the rapidity with which the pressure is lowered, as is the case with the flush. Both vary also with the amount of vaso-constriction of the artery, nice determinations being possible only with wide vessels. Martin, in observations on himself under different conditions, found differences of 5 to 19 mm. between the first and second flush, and quotes Schleiseik as placing the usual difference at 15 mm., Neu at 5 to 20 mm. in normal cases, and up to 45 mm. in pathological ones. These figures mean that, with too rapid lowering of the pressure, an underestimation of that extent would occur. Most observers also agree that, in a series of consecutive determinations by this method the later readings are invariably higher, to a greater degree than occurs with the Riva-Rocci method, and therefore not due wholly to a real reflex rise in aortic pressure. Stanton frequently found a variation of 20 mm. between the highest and lowest of a series of ten successive tests on the same patient. This is most easily explained by the development of a vaso-motor paresis in the finger, caused by continued compression.

---

[1] v. Recklinghausen, Martin, Stanton, Cook and Briggs, Hayaski, etc. Loc. cit.

In conditions of very low pressure the criterion fails entirely, and Gärtner himself attempts no measurements below 70 mm. During anæsthesia, also, and in toxic states, the method is difficult of application, and the observation of return of color is more inconvenient than the palpation of the pulse. Cook and Briggs gave up the use of the tonometer in part on this account.

The most serious fault of the criterion, however, lies in the fact that, at the best, it can only measure pressure in the small digital arteries. The extent to which these are subject to vaso-motor changes is notorious, and in this way variations in pressure within them may occur as a purely local manifestation. Cold affects them markedly, and all who work either with the Gärtner or the Mosso instruments lay special stress on the necessity for having the fingers warm. Brush and Fayerweather[1] found these peripheral changes so troublesome, with the latter instrument, that they modified it for use on the wrists. Wolf, in the series of seventy-two observations previously quoted, found differences between the readings in the two hands in one half the cases, the greatest being 25 mm. Hg. Such variation can only be due to defect in the method, or to local changes in the calibre of the digital arteries. Jellinek's[2] figures on the effects of exercise in a large number of healthy soldiers seem to me to illustrate this fault of the Gärtner method. Most of the men showed a rise in pressure, some no change, and a few a fall. One relation, however, was constant. Those with very low initial pressures always showed the most marked rise. Since we know how exercise tends to dilate the vessels of the skin, the explanation of his results, to my mind, is found in the probable marked vaso-constriction of the peripheral vessels which existed in his cases with low primary readings; this disappearing under exercise would allow the factitious original pressure to rise to the level of general blood-pressure.

---

[1] Brush, C. E., and Fayerweather, R. Observations on the Changes in Blood-pressure during Normal Sleep. Am. Jour. of Physiol., 1901, vol. v, p. 199.

[2] Jellinek, S. Ueber den Blutdruck des gesunden Menschen. Zeitschr. f. klin. Med., 1900, vol. xxxix, p. 457.

The use of this method of determining systolic pressure, then, has not the general applicability of those using the arm, nor have its results quite the same worth. Within these limitations it is of clinical value, is easily learned and, under favorable conditions, gives a beautifully distinct index of pressure, which lends it a certain charm.

c. Comparison of Systolic Pressures in Brachial and Digital Arteries.—Apart from local differences in vascular tone, the digital arteries undoubtedly have a slightly lower systolic pressure than the brachial, for the pulse wave in them is smaller. Most comparisons in the past have been with the Riva-Rocci instrument, using the narrow armlet, therefore with an overestimated brachial pressure. Hayaski, for instance, comparing the moment of disappearance of the radial pulse on one side, and the subjective throb on the other, found a fairly constant difference of 20 mm. in a large number of cases. As his normal persons were one hundred and eight school children, the elasticity error must have been small. v. Recklinghausen obtained an average reading from the finger 7 mm. Hg. lower than for the brachial.

B. Of Diastolic Pressure.—a. Maximum Pulsation of the Arterial Wall.—This means of determining the diastolic pressure within an artery, which we owe to Marey's keen insight, is unquestionably our most exact indirect measure of a blood-pressure. Mosso, when he first devised his sphygmomanometer, demonstrated its theoretical accuracy by the following experiment. Two vessels were taken, which communicated with one another by a vertical opening 40 mm. in diameter, closed by an unstretched elastic membrane. One vessel was connected with the pressure cylinder and recording manometer of his sphygmomanometer, the other with a mercury manometer, and with a pump used to imitate the heart in a model of the circulation. At the start of the experiment the pressure registered 76 mm. Hg. in each vessel. The membrane, therefore, was perfectly vertical. If the pressure was raised on either side, it was, of course, bulged out toward the opposite vessel. Now, while increasing the pressure on the side of the sphygmomanometer by successive steps, he produced plusation of the water in the other vessel, which was transmitted through the membrane to his recording manometer. The tracing shown in

Fig. 18 resulted. It is perfectly evident that the greatest pulsation is at 76 mm., the level of equal pressure on the two sides of the membrane.

Howell and Brush[1] have verified the principle for the artery itself. They used an apparatus devised by Walden, and shown in Fig. 19. The tube D was connected with a mercury manometer and with a Fick spring manometer, the latter magnifying and recording the pulsations. This is the general arrangement of Erlanger's sphygmomanometer. The left carotid of a dog was placed in this apparatus, and the right connected with a mercury manometer provided with maximum and minimum valves. The pressure in the closed cylinder was then raised 10 mm. at a time, and the pulsations at each pressure recorded by the Fick manometer. No difficulty was met in determining the greatest amplitude of the pulse-wave. The only source of error lay in the 10 mm. intervening between the readings, which made it possible that still larger waves might have occurred at some intermediate point. The error from this could

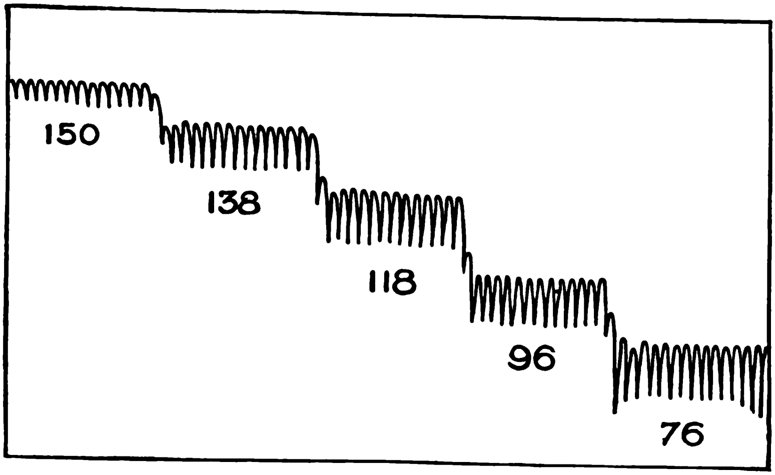


FIG. 18.—TRACING OF THE PULSATIONS TRANSMITTED THROUGH AN ELASTIC MEMBRANE SEPARATING TWO VESSELS.

(From Mosso, Arch. Ital. de Biol., 1895, vol. xxiii, p. 177.)

---

[1] Howell, W. H., and Brush, C. E., Jr. A Critical Note upon Clinical Methods of Measuring Blood-pressure. Boston Med. and Surg. Jour., 1901, vol. cxlv, p. 146.

not have exceeded 5 mm. Their results are shown in tabular form.

	Observation.	Diastolic pressure in right carotid measured by the minimum manometer.	Point at which the maximum pulsations were obtained in the left carotid as measured by the sphygmomanometer.
Experiment VII...	A	156.5 mm. Hg.	155 mm. Hg.
	B	156 " "	157 " "
Experiment VIII..	A	111 " "	112.5 " "
	B[1]	129 " "	129.5 " "
	C	82 " "	81 " "
	D	93.4 " "	85 " "
Experiment IX....	A	106 " "	109 " "
	B	110.5 " "	110 " "

[1] Animal bled profusely.

The measurements were practically simultaneous and under even conditions of anæsthesia, so that the pressure remained constant for considerable intervals. These figures, when coupled with Mosso's simpler demonstration, are convincing as to the essential accuracy of the point of maximum amplitude of pulsation as a guide to diastolic arterial pressure. Certain practical aspects of it alone remain to be touched on here.

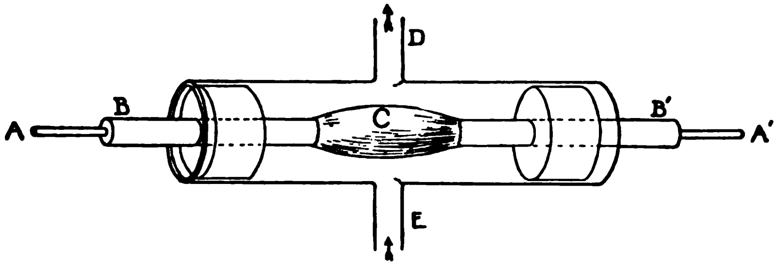


FIG. 19.—APPARATUS OF WALDEN FOR VERIFYING THE CRITERION OF MAXIMUM PULSATION OF THE ARTERIAL WALL.

AA′ = artery, tied, cut, and drawn through apparatus.
BB′ = small glass tubes inserted through stopper in larger tube.
C = tube of peritoneal membrane connecting the ends of BB′.
D = opening from outer tube leading to recording apparatus and manometer.
E = opening connected with pressure flask.

(From Howell and Brush.)

When used clinically, it is not possible to obtain thoroughly accurate judgments of where the pulsation is greatest, by simply watching the fluctuations of the mercury column. With a manometer of at least 3 mm. calibre, and rigid connecting tubing, quite large oscillations may be obtained, and a reasonable approximation of diastolic pressure made with a simple clinical instrument. With rapid pulses, neither the eye nor the mercury follow the movements quickly enough, and some form of tambour becomes necessary for magnifying and recording them. For this reason the types of apparatus which give diastolic pressure satisfactorily in all cases are more bulky, require longer for a reading, and are more costly, than the simpler ones measuring systolic pressure. Hill and Barnard's is an exception as regards the first two drawbacks; but its delicate metal tambour is easily disordered, and does not give exact readings unless frequently corrected by comparison with a mercury manometer. Besides, it is not a recording manometer, and sometimes gives one trouble in determining the maximum of the oscillations. It must also be borne in mind that the pulsations, when once they have attained their maximum with increasing pressure, remain at much the same height for a number of millimeters, then decline rather suddenly as systolic pressure is approached. Hill, who thought his method indicated mean arterial pressure, advised taking the middle point between the limits of maximal pulsation. This may at times correspond with mean pressure, but is not at all an exact guide.

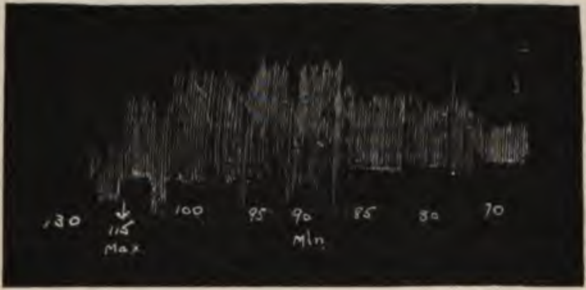


FIG. 20.—TRACING FROM A NORMAL PULSE BY ERLANGER'S SPHYGMOMANOMETER.

At 115 mm. is seen the abrupt increase in amplitude which indicates systolic (max.) pressure. At 90 mm. the pulsation is still maximal, but at 85 mm. it is much diminished. 90 mm. is therefore the diastolic (min.) pressure.

The correct manner of using the criterion, to whatever instrument it be applied, is to lower slowly the pressure from the systolic level, if possible only 5 mm. at a time, watching the size of the pulsations. The lowest pressure at which they are still of maximal height is the diastolic pressure. Below this they decrease very rapidly in amplitude, so that the determination seldom offers special difficulty with a graphic record. The accompanying tracing (Fig. 20), taken with the Erlanger sphygmomanometer, illustrates the method. With irregular pulses and when rapid fluctuations of pressure are present, as in forced breathing, the criterion fails.

C. Other Criteria, not in Clinical Use.—Hürthle[1] rendered the arm bloodless with an Esmarch bandage, then placed it in a plethysmograph cylinder filled with water and connected with a manometer. When the compressing band was removed, the return of blood to the arm displaced water from the cylinder, and automatically registered in the manometer pressures, from which he thought the maximum, minimum, and mean arterial pressures could be read. The results are somewhat questionable, the application abounds with opportunity for subjective mistakes, if the reading does not, and the apparatus is impossible outside the laboratory.

v. Recklinghausen made his determinations by recording the pulse from the armlet during steadily increasing compression, by means of a Fick manometer and revolving drum. His manometer was standardized, and, by comparing the portions of the "staircase curve" so obtained, he determined the exact value of each portion of the pulse-wave, from this reconstructing a pulse-wave in its exact measurement expressed in pressures. This, also, is obviously unsuited to general imitation.

Hensen,[2] Masing,[3] and the author,[4] before any satisfactory clinical method for determining diastolic pressure had been brought forward, endeavored to investigate it by a combination of sphygmographic tracings and the Riva-Rocci apparatus. Masing and myself used an identical method, independently of one another, which gave merely approximations and could in no way be recommended to-day.

---

[1] Hürthle, K. Ueber eine Methode zur Registierung des Arteriellen Blutdruck beim Menschen. Deutsch. med. Wochenschr., 1896, p. 574.

[2] Hensen, H. Beiträge zur Physiologie und Pathologie des Blutdrucks. Deutsch. Arch. f. klin. Med., 1900, vol. lxvii, p. 438.

[3] Masing, Ernst. Ueber das Verhalten des Blutdrucks des jungen und des bejahrten Menschen bei Muskelarbeit. Deutsch. Arch. f. klin. Med., 1902, vol. lxxiv, p. 253.

[4] Janeway, Theodore C. Some Observations on the Estimation of Blood-pressure in Man, with Especial Reference to the Results obtained with the Newer Sphygmomanometers. N. Y. Univ. Bull. of the Med. Sci., 1901, vol. i, p. 105.

CHAPTER IV

THE MODERN SPHYGMOMANOMETERS

1. Introduction.

2. Instruments measuring systolic pressure only.

A. Criterion: The return of the pulse-wave under compression.

- a. General construction.
- b. General method of use.
- c. Riva-Rocci's sphygmomanometer.
  - Special construction and technique.
  - Advantages and defects.
- d. Martin's modification with Gumprecht's manometer.
- e. Cook's modification.
  - Special construction and technique.
  - Advantages and defects.

B. Criterion: The return of color to the skin of the blanched finger.

- a. Gärtner's tonometer.
  - Special construction.
  - Method of use.
  - Advantages and defects.
- b. Martin's modification.

3. Instruments measuring diastolic pressure.

A. Criterion: Lowest level of maximum pulsation.

- a. Hill and Barnard's sphygmometer.
  - Construction.
  - Method of use.
  - Advantages and defects.

4. **Instruments measuring systolic pressure** (return of the pulse), **and affording an approximate estimate of diastolic pressure** (maximum pulsation).

   a. Stanton's sphygmomanometer.
      Special construction and technique.
      Advantages and defects.

   b. Author's sphygmomanometer.
      Special construction and technique.
      Advantages and defects.

5. **Instruments measuring accurately both systolic pressure** (return of the pulse) **and diastolic pressure** (maximum pulsation) **by graphic record.**

   a. Erlanger's sphygmomanometer.
      Special construction.
      Method of use.
      Advantages and defects.

6. **Comparison of results obtained with the various sphygmomanometers.**

7. **Blood-pressure records.**

8. **Practical suggestions.**

   A. The choice of a sphygmomanometer.
   B. Minor details.

CHAPTER IV

THE MODERN SPHYGMOMANOMETERS

1. INTRODUCTION

THE gradual development of various sphygmomanometers from which one may choose a clinical instrument to-day, has been unfortunate in breeding more partisan bias and personal feeling than should find a place in the quest of scientific accuracy; but this evil has not been without its good side. It has led to the rigid scrutiny of each new instrument brought forward, and a diligent search for its faults. The result, we have seen, has been to leave the method of circular compression in full possession of the field. When complying with the requirement that the compressing surface shall bear a definite proportion to the circumference of the part to which it is applied, the error involved is negligible for clinical purposes. The values so obtained for the blood-pressure are comparable from individual to individual, and are almost entirely independent of differences in the soft parts overlying the vessel. An armlet of 12 cm. width is adequate for any but the most enormous arms.

The investigations which have led to this conclusion have been of recent date, and practically all our data relating to human blood-pressure are from instruments using a narrower compressing surface. These will be described severally in the latest form in which they are purchasable, but no instrument with a narrow armlet should be used. One may easily substitute a wide cuff in any of the forms described.

2. INSTRUMENTS MEASURING SYSTOLIC END-PRESSURE ONLY

A. Criterion: The Return of the Pulse-wave under Compression (see page 62).—For all instruments the general construction and method of use are the same.

a. General Construction.—ESSENTIAL PARTS: A, manometer; B, compressing armlet; C, inflating apparatus.

ACCESSORY PARTS: D, connecting tubing with air-tight joints; E, valve or pinch-cock for the slow release of pressure.

b. General Method of Use.—The armlet is fastened snugly (see page 56) about the arm or thigh at the level of the heart (see page 59). On the thigh this necessitates the recumbent posture. The finger ordinarily used for palpating the pulse is then placed on the radial, or the dorsalis pedis, and the patient instructed to relax the limb completely (see page 61). With the other hand the pressure is then raised steadily by means of the inflating apparatus. When the volume of the pulse begins to diminish, the pressure is increased very slowly until the pulse disappears, then released as gradually as possible until its return is detected. The height of the manometer column at which this occurs is noted, and the pressure raised just enough to again obliterate the pulse. It is allowed to fall a second time and another reading made. If the two points correspond within 5 mm. of one another, the average may be taken as the systolic blood-pressure at the time of observation. If the readings fail to approximate so nearly, one must wait a couple of minutes and determine anew.

c. Riva-Rocci's Sphygmomanometer.[1]—SPECIAL CONSTRUCTION.—A. The mercury manometer is of cistern form. The tube carries behind it a metal scale graduated in millimeters from 40 to 260 mm. The cistern is of heavy glass with two tubes entering it, one for the attachment of the inflating bulb, the other for the tube leading to the armlet. The latter of these is provided with a screw-valve (E) for the gradual lowering of pressure. The whole stands on a solid metal base.

B. The armlet consists of a hollow rubber tube covered with silk, of 4½ cm. diameter. It is provided with a special clamp for fastening in place on the arm. Near its closed end is inserted a small tube (D) leading to the manometer.

C C′. The inflating apparatus is a Richardson double bulb, such as used for a thermo-cautery.

SPECIAL TECHNIQUE.—The armlet is placed about the arm and the open end secured firmly in the clamp. The pressure is best raised by means of the terminal bulb (C) until the pulse has nearly disappeared, then the final gradual compression and release regulated from the distended second bulb (C′). This requires some practice and, for high pressure, a little strength, but it saves enough time to be worth acquiring. If not possible, then the slow lowering of pressure must be accomplished with the screw-valve (E).

---

[1] Riva-Rocci. Gaz. Med. di Torino, 1896, Nos. 51, 52.

ADVANTAGES.—It is the original instrument. The manometer stands firmly and the scale is easily read. It is not easily broken. The armlet can be neatly adjusted. The double bulb inflation requires no special knack.

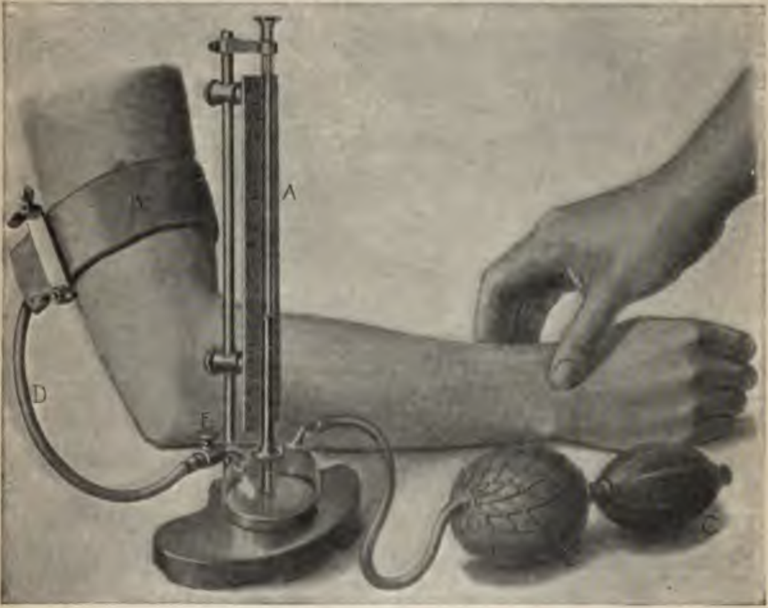


FIG. 21.—RIVA-ROCCI'S SPHYGMOMANOMETER.

DEFECTS.—The manometer is not easily portable. The armlet possesses the fatal defect of too small width (see page 57). The double bulb as an inflator consumes an unnecessary amount of time and is not very durable. The connecting tubes are of distensible rubber.

MAKERS.—Zambelli & Co., Turin, Italy.

COST.—In Germany, 45 marks. Can be obtained in the United States only by importation.

d. Martin's[1] Modification with Gumprecht's Manometer.—This substitutes a manometer with glass cistern of larger capacity, in order to make the zero point accurate; also a 10 cm. armlet with an improved clamp. The manometer, armlet, bulb, and an outlet tube closed by a pinch-cock are connected through a 4-way glass tube.

This overcomes the chief defects of the original instrument, but the manometer is not easily portable.

MAKERS.—Gehrike, Jena, Germany.

e. Cook's Modification.[2]—This is the most widely used instrument in this country and has done most to arouse interest in the study of blood-pressure.

SPECIAL CONSTRUCTION.—A. An ingenious portable manometer with jointed tube; calibre 1 mm.

B. A simple and cheap armlet, consisting of a narrow rubber bag, 4½ by 40 cm., covered with canvas and fastening with hook and eye under a band.

C C′. Richardson's double bulb for inflation.

D. A glass T canula joining the connecting tubes from manometer, armlet, and bulb.

E. A pinch-cock on a small branch tube for the release of pressure. This is introduced near the manometer by means of a second glass T canula.

SPECIAL TECHNIQUE.—This is practically the same as for the Riva-Rocci, except that the armlet is fastened by passing the open end under the band on the closed end and hooking it into one of the series of eyelets. Care must be used to see that the upper joint of the manometer tube is inserted securely. For those who are unable to control the pressure from the second bulb (C′), a very gentle squeezing of the pinch-cock will allow slow escape of the air. In packing the instrument, this pinch-cock must be placed on the tube connected with the neck of the manometer reservoir, or mercury will be spilled.

ADVANTAGES.—It is exceedingly simple, light and compact; therefore well fitted for general clinical use.

DEFECTS.—The manometer does not stand firmly and is readily broken, at least in my experience. Without care in packing, the mercury is apt to be spilled. The scale is not easily read and the tube calibre is too small. The connections are made with distensible rubber tubing. All these defects would be compensated for by its simplicity and compactness, but it possesses, like its original model, the serious defect of providing much too narrow an armlet (see page 57). Its readings, therefore, tend to be too high in all adults, much too high in stout ones. In children this does not enter into the consideration, and the instrument was first used by Dr. Cook in a children's hospital. For comparative readings on the same patient the error will be constant and negligible, thus it may be used in surgical operations and acute disease for following variations in tension, but its results are not trustworthy for diagnosis.

---

[1] Martin, Alfred. Münch. med. Wochenschr., 1903, L[1], pp. 1021 and 1072.

[2] Cook, H. W. Blood-pressure Determinations in General Practice: Introducing a Practical Instrument for Routine Use. Jour. Am. Med. Ass., 1903, vol. xl, p. 1199.

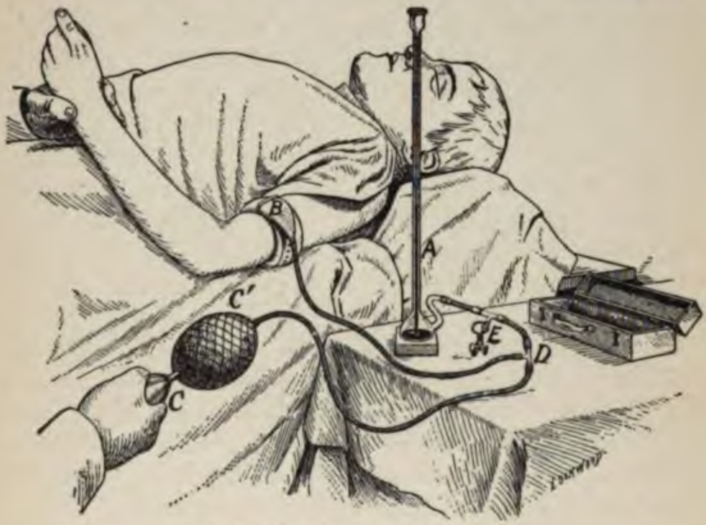


Fig. 22.—Cook's sphygmomanometer.
(From Cook and Briggs.)

Makers.—Eimer and Amend, 205–211 Third Avenue, New York city.

Cost.—Complete in case, $8.50 net.

They also make a form with one piece manometer for hospital use, where portability is unnecessary. Cost, $6.50 net.

An instrument practically identical with Cook's hospital form, except that the manometer is modelled after Gartner's, and has a more legible scale and broader foot, is made by The F. H. Thomas Co., 707 Boylston Street, Boston, Mass. This also is sold at $6.50 net. It is not portable.

B. Criterion: The return of color to the skin of the blanched finger (see page 66). **a. Gärtner's Tonometer.**[1]—This is the only instrument using the above criterion. Introduced by Prof. Gärtner in the summer of 1899, it has achieved a wide popularity. In its essential parts it does not differ from the instruments of the Riva-Rocci type, save that the circular compression is applied by a ring to the finger. The plate shows its construction.

SPECIAL CONSTRUCTION.—A. The mercury manometer, of simple cistern form, stands firmly on a broad foot. The pasteboard scale, graduated in 0.5 cm. divisions from 0 to 26 cm., is readily seen at a distance. It is not easily carried, and for this purpose a metal manometer is furnished, which cannot be trusted without frequent standardizing.

B. The pneumatic ring, the original feature of the apparatus, consists in a metal ring 1.5 cm. in width, lined with a rubber membrane which leaves a hollow, but air-tight space between it and the metal. At one point the outer ring is pierced by a metal tube, through which the interior air space is connected with a tube leading to the manometer and inflator.

C. A rubber ball or bulb, without valve, is used for inflating. The slow reduction of pressure is insured by placing the ball between the jaws of a simple vice (E). This is allowed to open slowly by turning the nut which runs on a screw thread. The bulb is put in circuit with the manometer and ring by stiff tubing and a T canula (D).

F. A compressor, by means of which the finger can be rendered bloodless, is the remaining accessory. It consists of a rubber membrane stretched over the head of a small metal cylinder.

METHOD OF USE.—A pneumatic ring of suitable size is placed on the second phalanx of the middle or ring finger, which must be at the level of the heart and must also be perfectly warm. The last phalanx is then rendered bloodless by squeezing the compressor over it. This is held in place until the pressure in the ring is raised above the expected systolic blood-pressure by means of the bulb. The compressor is then removed from the finger-tip and the vice which holds the bulb allowed to open slowly. As it does, the air gradually returns from the ring into the bulb and the pressure in the whole system falls. The finger-tip is watched intently and, at a certain point, it is seen to flush suddenly; then the height of the manometer column is taken. At about the same time the patient is conscious of a throb. In some cases, if the pressure be reduced very gently, the throb will be a few mm. Hg. lower than the first flush and will be accompanied by a deepening of it. The manometer reading at the instant the color returns represents, with the reservations made heretofore (see page 66), the systolic end-pressure in the digital arteries.

---

[1] Gärtner, G. Wien. med. Wochenschr., 1899, vol. xlix, p. 1412.

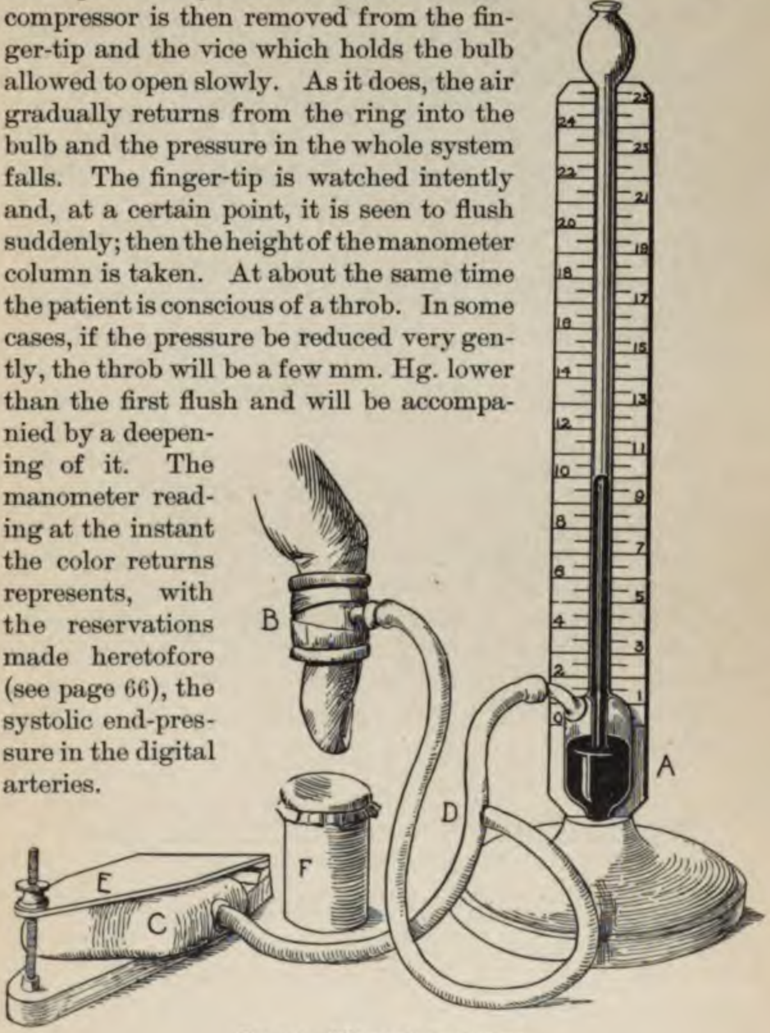


FIG. 23.—GÄRTNER'S TONOMETER.

ADVANTAGES.—In most cases the easy perception of the return of color to the finger gives this method a strong recommendation. It requires no special skill in its application.

Defects.—Unfortunately these are numerous enough to outweigh the advantages, and the majority of careful observers, in this country as abroad, have adopted methods which can be applied to the arm. These defects are due in part to badly fitting rings (see page 56), unless one have a large assortment of sizes, or to careless application (see page 60); in part to insufficient width of the compressing surface (see page 59). Most important of all, and least remediable, are the errors inherent in all methods which measure blood-pressure in arteries so small and so near the periphery as the digitals (see page 68), and the failure of the criterion to work in a certain number of patients.

The instrument is not sufficiently portable for all purposes and is used with difficulty on unconscious, delirious or anæsthetized patients.

Maker.—Franz Hugershoff, Leipzig, Carolinenstrasse 13, Germany.

Cost.—25 marks 50 pfennig. With metal manometer, 35 marks. At times may be found at instrument dealers in the United States without special importation.

b. Martin's modification.—This consists only in the substitution of a small modified Riva-Rocci tube, of 2.5 cm. width, for the pneumatic ring. Martin[1] has devised a special clamp for this and has eliminated the technical defects of the instrument, but not the errors of the method.

These rings may be had of Hanhart & Co., Zurich, Switzerland.

3. INSTRUMENTS MEASURING DIASTOLIC PRESSURE

A. Criterion: Lowest level of maximum pulsation (see page 69). **a. Hill and Barnard's Sphygmometer.**[2]—This is the sole instrument of this class which can be considered a modern sphygmomanometer, Oliver's hæmodynamometer employing the faulty v. Basch method of compressing the artery. The important parts, except for the type of manometer used, are analogous with those of the Riva-Rocci, and are seen in the cut.

---

[1] Martin, Alfred. Münch. med. Wochenschr., 1903, L^1, p. 1072.

[2] Hill, Leonard, and Barnard, Harold. British Med. Jour., 1897, vol. ii, p. 904.

Special Construction.—A. A delicate metal manometer or spring tambour. The dial is graduated in recent instruments from 30 to 250 mm. Hg., each subdivision representing 2 mm. The needle magnifies considerably the oscillations of the pulse-wave. The stem of the manometer has a lateral branch, to which the tube leading from the armlet is attached by a screw-cap connection; and a straight branch, to which the pump is attached.

B. The armlet consists of a hollow rubber bag, 5 cm. wide and about 20 cm. long, with the connecting tube (D) cemented into its centre. It is attached to an outer leather cuff which buckles around the arm.

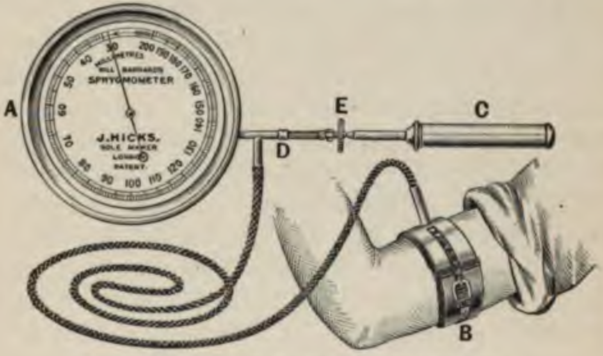


Fig. 24.—Hill and Barnard's sphygmometer.

C. The inflating apparatus is a pump, like a hand bicycle pump. At E is a valve for the slow escape of the air.

Method of Use.—The cuff is buckled snugly around the arm, with the same precautions as for the Riva-Rocci armlet, and with the outlet tube directed anteriorly. The pressure is then raised slowly and steadily with the pump. Soon the needle will be seen to pulsate. Its oscillations will increase until they cover a number of millimeters of the scale, then gradually decrease as the pressure is further raised. The manometer reading at which they first become most extensive is taken. The pressure is then allowed to fall slowly by turning the wheel which releases the valve, and the last point of greatest oscillation of the needle is noted on the descent. This should correspond closely with the previous reading and their average represents the diastolic pressure. Hill recommended taking the mid-point of maximum oscillation, which he thought corresponded with mean blood-pressure, but this is an indefinite guide (see page 69), and the method I have given should be followed.

ADVANTAGES.—The construction and finish of the instrument are admirable; it is easily carried in its leather case, is not difficult to use, and seemed at first an almost ideal clinical method.

DEFECTS.—Like all delicate metal manometers, the readings tend to become inaccurate. One which I used during 1900 and 1901, registered 26 mm. too high at the end of a year. It needs to be standardized frequently by comparison with a mercury manometer, which is irksome. The manometer is also difficult of repair in this country, a considerable drawback to so costly an apparatus. It has the narrow cuff, with its inherent errors; though a wide one might of course be substituted for it. In addition, in certain important cases, such as aortic insufficiency and high tension, the determination of the exact lower limit of maximal oscillation is not always easy, and I am inclined to the view that, for clinical purposes, the knowledge of diastolic pressure alone is not so valuable as that of systolic. The latter may be determined with this instrument, as with the Riva-Rocci, by the disappearance or return of the pulse; but the upper limit of use, 240 mm., has not sufficed for many cases in my experience. The statement made by some who use it, that systolic pressure corresponds with the point at which the needle no longer shows movement, is quite mistaken. Marey, in his original work on the subject (see page 50), pointed this out, and Howell and Brush[1] have demonstrated it conclusively. With a maximum manometer in the artery, they found the pulse-wave carried over to the sphygmomanometer as much as 20 mm. beyond the level of maximum pressure. This undoubtedly is due to the fact that only the middle of the portion of artery under the bag is compressed, the upper part still remaining open and the pulse in it striking against the armlet.[1]

---

[1] Howell and Brush. Boston Med. and Surg. Jour., 1901, vol. cxlv, p. 146.

MAKERS.—J. Hicks, London, Eng.; agents for the United States, Oelschlager Bros., 42 East Twenty-third Street, New York city.

COST.—In the United States, $40.

4. INSTRUMENTS MEASURING SYSTOLIC PRESSURE (Return of the Pulse) AND AFFORDING AN APPROXIMATE ESTIMATE OF DIASTOLIC PRESSURE (Maximum Pulsation)

By using an armlet whose expansion outward is entirely hindered by a stiff cuff, connected with a wide calibre manometer by stiff-walled tubing, the pulse-wave can be transmitted to the mercury column. The latter does not, of course, follow the cardiac variations in arterial pressure quantitatively (see page 5), but the movements of the mercury are of sufficient extent to make an approximate reading of the lower limit of maximum oscillation possible. With rapid and small pulses this becomes difficult or impossible, but in the average case the column fluctuates 6 mm. to 10 mm. at its greatest. In pulses of high tension and in aortic insufficiency, where a knowledge of diastolic as well as systolic pressure is especially important, the column may show, as in a case I observed recently, a pulse movement of as much as 40 mm.

Two instruments recently brought forward in this country have the requisite construction for this purpose.

A. Stanton's Sphygmomanometer.[2]—SPECIAL CONSTRUCTION.—Stanton was the first in this country to adopt the wider armlet and to construct a clinical instrument free from serious defect. His sphygmomanometer differs from the Riva-Rocci chiefly in the compressing and inflating devices. The former (F) is a hollow rubber bag, 3¼ inches (8 cm.) wide and 16 inches long, closed at both ends, and prevented from expanding outward by a cuff of double thick canvas, reinforced by tin strips. A glass connecting tube is inserted at the centre of the rubber bag by means of a hollow valve stem, such as is used for bicycle tires. This is connected by the stiff-walled rubber tube (G) with the manometer, which consists of a metal cistern (C) connected by a metal tube with the glass upright tube and scale (D), which can be unscrewed for carrying. The relative diameters of cistern and column are 100 to 1, thus affording an accurate zero point. The cistern (C) is covered by a screw-cap with a T-tube, one branch of which connects with the armlet, the other with the bulb. At B is a screw valve for gradual release, at A a stop-cock to shut off the inflator. Stanton uses a single rubber bulb, such as is found on atomizers, for the latter.

---

[1] See diagram page 57. The portion of artery A B C would not be completely collapsed.

[2] Stanton, W. B. Univ. of Penn. Med. Bull., 1903, vol. xv, p. 466.

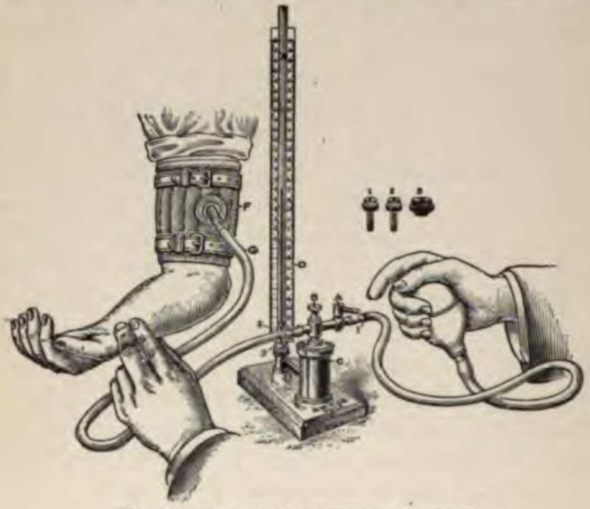


FIG. 25.—STANTON'S SPHYGMOMANOMETER.

(Cut from The Arthur H. Thomas Co.)

To carry the apparatus, the bulb is removed from the T at 1 and the cap 1 screwed in, cap 2 similarly closing the other entrance 2, from which the armlet and tube G are disconnected. The manometer is then tilted on its side until the mercury has all run into the cistern, the upright glass tube is unscrewed, and its place filled by the cap 3.

SPECIAL TECHNIQUE.—The methods of estimating systolic and diastolic pressures are the same as those described for the Riva-Rocci and Hill and Barnard instruments respectively.

The armlet is applied snugly, folding in any extra length of the rubber cuff, if the arm be small. The manometer column should be inspected to see that it stands at 0, the armlet then connected with the manometer, the valve (B) closed, and the stop-cock (A) opened. The bulb is squeezed as shown until the pulse disappears, when the stop-cock (A) is closed. The pressure is gradually lowered by unscrewing B, noting the return of the pulse and the lowest point at which maximum pulsation of the mercury occurs. The precautions necessary in noting the oscillation of the mercury will be explained in connection with the author's sphygmomanometer.

ADVANTAGES.—The greatest recommendation of this instrument is the fact of its throwing light on the diastolic pressure, while measuring the systolic with the same ease as the ordinary forms of the Riva-Rocci. Of course it does not give measurements which compare with those of Erlanger's graphic method, or Mosso's, but the latter are unquestionably too bulky and too elaborate for general use. This sphygmomanometer also has an armlet wide enough for the majority of cases, though not so absolutely free from error as a 12 cm. one.

DEFECTS.—It seems to me unfortunate not to adopt the full 12 cm. armlet, for on arms of over 30 cm., and they are not very rare, some error must be present. I must admit the awkwardness of the wider cuff, but this should be a minor consideration. Beyond this, the instrument possesses no defect, save that it is not quite so readily portable as those devised by Cook and by the author. It is an excellent and ingenious clinical instrument.

MAKERS.—The Arthur H. Thomas Co., southwest corner Twelfth and Walnut Streets, Philadelphia, Pa.

COST.—Complete in carrying case, $20.

B. The Author's Sphygmomanometer.—SPECIAL CONSTRUCTION.—This instrument was devised by me after much hesitation, for I had no wish to increase the number of existing sphygmomanometers. It was the result of a desire to possess a really portable clinical instrument in which, nevertheless, no essential accuracy should be sacrificed. The only original feature is the folding U tube manometer (A). This is shown in the plate in position for use. For carrying, the upper joint of the manometer tube is removed and slipped through the rings to the right. The open end of the U is then closed by a small cork (F); the other end is closed automatically when the case is shut, by a block which compresses the rubber joint (G). The scale is slid down, the Politzer bag (C) removed from the stop-cock (E), which contains a needle-valve for slow release of pressure. This stop-cock is allowed to slip under a

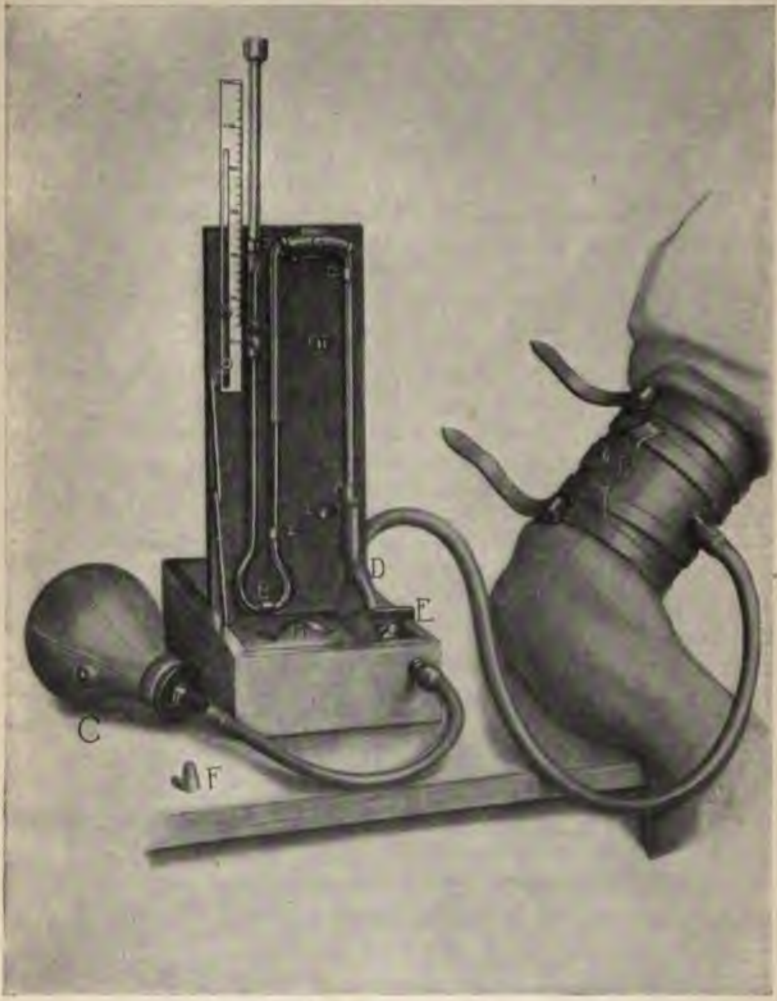


FIG. 26.—AUTHOR'S SPHYGMOMANOMETER.

spring (H), as the case closes. The lid, to the under side of which the manometer is fastened, is then closed by dropping the catches which hold it behind and folding down the hinge at the left, the lower end of the lid sliding back in a groove. The whole, when closed, measures 10¼ × 4⅝ × 1⅞ inches and, with the armlet and inflator, weighs 2½ pounds. The manometer tube has a calibre of 3 mm. and all the connections are of heavy pressure tubing. The armlet (B) is a hollow rubber bag, 12 cm. wide by 18 cm. long, with an outer leather cuff 15 × 33 cm., fastened, as the illustration shows, by two straps with friction buckles which will catch at any point. The buckle employed makes adjustment to different arms much easier than the ordinary tongue buckle. Experience with the latter has taught me that one hole may be too tight and the next too loose. For the inflating mechanism I have followed Erlanger in adopting the ordinary Politzer bag, though I find one with the valve necessary to fill the 12 cm. armlet. The needle-valve is manipulated with ease, even by a novice, and allows a very gradual or a sudden lowering of pressure equally well.

As accurate manometer tubes cannot be made in this country, each scale is graduated empirically, the reading at each point being the exact difference between the level of the two columns of mercury. The instruments are therefore accurate.

SPECIAL TECHNIQUE.—The procedure in general is the same as with Stanton's instrument. The armlet is buckled on, overlapping the ends if the arm be small. The outlet tube should be directed anteriorly. The scale of the manometer is then set with the zero point at the level of the two mercury columns, and the security of the upper joint of the tube tested. The finger ordinarily used for palpating the pulse is then placed on the artery at the wrist. If the left hand is employed for this, the left arm of the patient should be used. The other hand grasps the Politzer bag firmly and raises the pressure until the pulse is obliterated, then releases it very slowly and steadily until it returns. This gives the systolic pressure. With high tension pulses the Politzer bag may not contain air enough to raise the pressure to the point of obliteration, as the 12 cm. armlet has a considerable cubic volume. If this is found, close the stop-cock (E) while the Politzer refills, then open slowly while squeezing the Politzer, and the pressure can be carried as high as desired. Then close the stop-cock, place the finger on the pulse, and with the other hand open the cock slowly until the air begins to escape through the needle-valve. In this way lower the pressure very gradually until the return of the pulse is detected. Then carry the pressure a little higher and repeat. This time, after the pulse is detected, allow the pressure to fall 5 or 10 mm. at a time until the lowest point of maximum oscillation of the manometer column is determined. If any air remains in the apparatus, remove the Politzer bag and it will escape. One precaution must be observed, namely, to allow time after each fall in pressure for the mercury to recover from the procedure, before comparing its oscillations with those at the previous level. If the drop has been sudden there will be a rebound of the column, which will make the first few pulsations abnormally large. The respiratory variations in size of the pulse are quite evident in some patients, and at least ten to twenty oscillations should be observed at each level to obtain an average. I think it usually wise to make a rough approximation first, by allowing the pressure to fall 10 mm. at a time; then, after releasing all the pressure, to return to just above the point thus determined and make a careful estimation at 5 mm. intervals. When the greatest fluctuation of the column does not exceed 5 mm., it is not possible to form any judgment, and the same holds good when the pulse is very rapid. Too small amplitude of pulsation is a direct effect of a loose adjustment of the cuff, and this should be inspected before giving up the attempt to find the diastolic pressure.

ADVANTAGES AND DEFECTS.—These will perhaps be brought out more clearly in the hands of others. As a portable sphygmomanometer it is second only to Cook's, which has not proved substantial in my hands or those of my hospital internes, and which also lacks the wide armlet. It, and Stanton's form, are the only simple instruments with which one may approximate diastolic pressure, besides measuring systolic. It seems to me more compact and portable than the latter, as well as less complicated and expensive. I might add that no attempt has been made to have the case contain the non-breakable armlet and inflator, which would add materially to the weight. These, with the manometer, will fit in the bottom of any common hand-bag, with one's other instruments.

Makers.—Charles E. Dressler & Bro., 145 East Twenty-third Street, New York city.

Cost.—Complete, $14.00.

For my hospital use I have designed a similar instrument, but with one-piece manometer, and a box which does not close. Mr. Dressler can make these to order.

For physiological instruction in medical schools, the armlet and inflator can easily be attached to the regular laboratory[1] manometers.

5. INSTRUMENTS MEASURING ACCURATELY BOTH SYSTOLIC PRESSURE (Return of the Pulse) AND DIASTOLIC PRESSURE (Maximum Pulsation) BY GRAPHIC RECORD.

Erlanger's Sphygmomanometer.[1]—In its improved form this is one of the latest, and certainly is the most accurate addition to our means of determining human blood-pressures. It employs the same criteria as the previous instruments, but the return of the pulse and the point of maximum pulsation are both made so clearly visible, that the subjective errors are minimized.

Special Construction.—The mechanism of this instrument is more complicated than of those previously described, and thoroughly original, but the only essential difference is the addition of a recording device. Fig. 27 shows the whole apparatus in perspective. The U-tube manometer connects with a 4-way tube, one branch of which leads to the armlet, another (F) to the special stop-cock (C D), which will be described later, and through it to the inflator. The vertical branch (to P S) communicates with the interior of a rubber bulb (B), enclosed in a heavy glass bulb (G), like Marey's sphygmoscope, which, under certain conditions, opens freely to the atmosphere through the tube E. The object of this device is to shield the delicate tambour from too sudden changes of pressure, which

---

[1] Erlanger, Joseph. Am. Jour. of Physiol., 1904, vol. x, Proceed. of Am. Physiol. Soc., p. xiv.

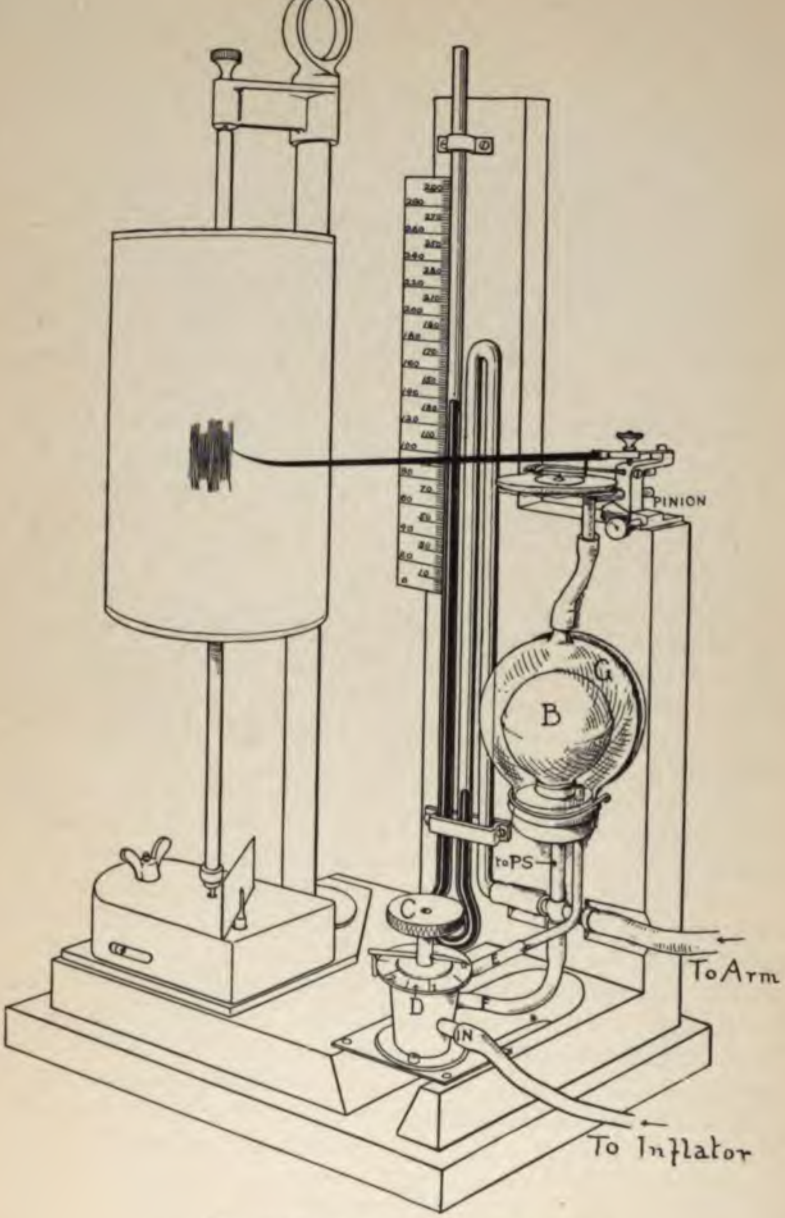


FIG. 27.—ERLANGER'S SPHYGMOMANOMETER.

might rupture the diaphragm. The tambour is connected with the air enclosed in the glass bulb (G), and inscribes its pulsations by an aluminum lever, as an ample tracing on the revolving drum. The pinion designated in the figure allows the tambour to be rotated, so that the lever may be brought to bear on the drum, or removed from it. The tracing is made on smoked paper, instead of black on white, as the figure, on account of the technical limitations of a line drawing, represents it. The drum has two speeds, the faster obtained by removing the fan, and is controlled by a lever, which projects through the slot on the face of the box covering the clockwork. The base and back are of wood, and a wooden cover is provided for carrying, with a handle on its top. The whole apparatus is considerably larger than an ordinary microscope and case, and of about the same weight.

The compressing armlet is a rubber bag, 12 by 16 cm., with an outer leather cuff, fastening by three buckles. A Politzer bag is used for inflation. All rubber tubing is the heaviest obtainable, so that no loss of the pulse-wave occurs during transmission.

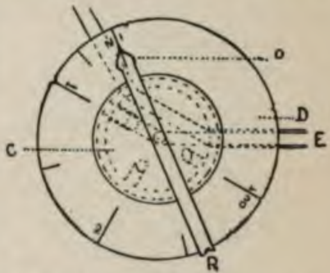


Fig. 28.—Diagrammatic view of stop-cock from above.

(From Dr. Erlanger's drawing.)

The details of the stop-cock and recording mechanism may be more easily explained by reference to the mechanical drawings, kindly loaned me by Dr. Erlanger. The former, in which the whole technique of use centres, is the most ingenious feature of the apparatus. It consists of a brass plug C, fitting in a barrel which is surmounted by a dial D. Fig. 28 shows this from above. The head of the plug (C) carries a pointer (P), the shaft of which terminates at its opposite end in a pawl (R). This pawl catches lightly, with a click, like the triple nosepiece of a microscope, in certain grooves on the dial. The grooves mark important positions of the stop-cock, and are designated "In," "Out," 1 and 2. At any intermediate point, no connection exists through the stop-cock, and the manometer, tambour, and armlet make a closed circuit, the tambour then registering the pulsation in the arm, and the manometer the absolute height of pressure in the whole system. The minute opening (O) in the tambour, prevents suction on the diaphragm during slow release of the pressure, but it is too small to influence the trace caused by the rapid pressure variations of the pulse-wave. When the pointer is set at "In," as in Fig. 28, the stop-cock makes the connection shown in Fig. 29.

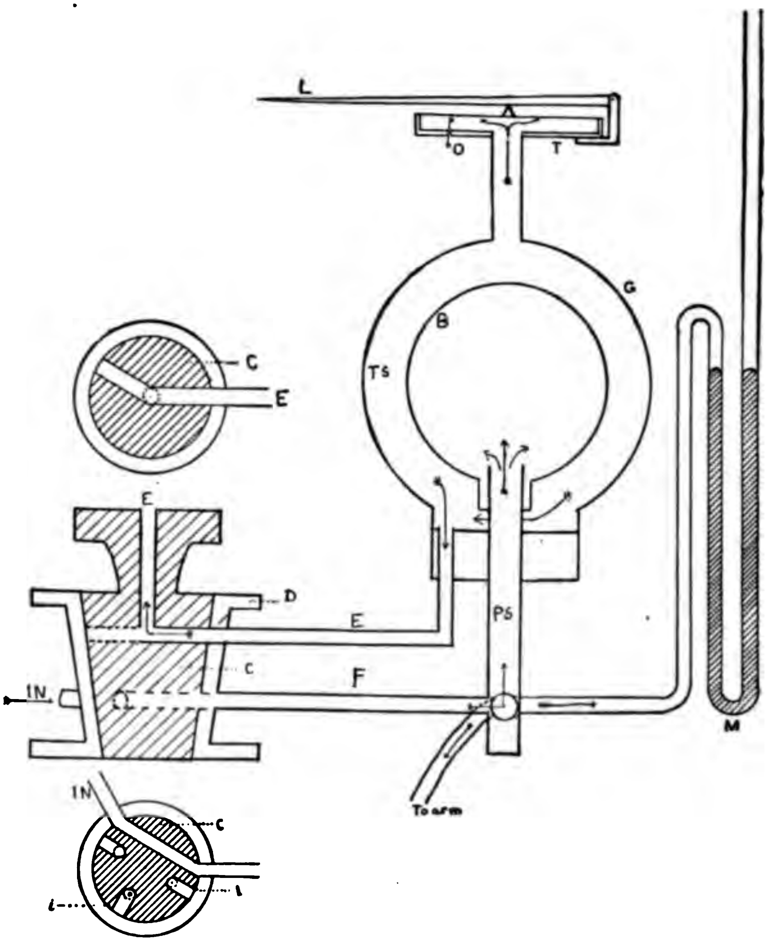


FIG. 29.—DIAGRAMMATIC SECTION OF ERLANGER'S SPHYGMOMANOMETER. STOP-COCK AT POSITION "IN."

Above and below the vertical median section of stop-cock are horizontal sections, the upper at level of tube E, the lower at level of tube F. (From Dr. Erlanger's drawing.)

The manometer, armlet, and pressure space (P S), inside the rubber bulb (B), are in circuit, through the tube (F), with the inflator attached to the tube "In." The air within the glass bulb (G) which communicates with the tambour, marked T S (tambour space), is open freely to the exterior through the tube (E), which connects with a vertical bore in the center of the stop-cock (C). Thus, as the pressure in the apparatus is raised and the bulb (B) expands, air is displaced out of T S, and the lever of the tambour does not change its position, except for a moment. By a slight turn of the stop-cock all these connections are broken, and the lever (L) begins to write, for the pulsations of the arm are reproduced by the bulb (B), and the air forced in and out of T S can only go to the tambour.

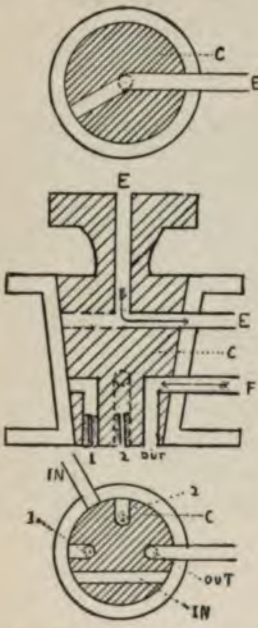


Fig. 30.—Diagrammatic sections of stop-cock, as in Fig. 29. Position "OUT." (From Dr. Erlanger's drawing.)

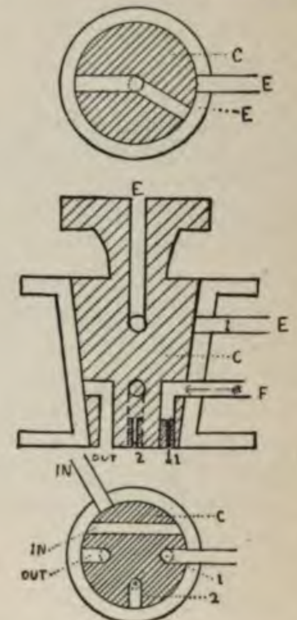


Fig. 31.—Diagrammatic sections of stop-cock, as in Fig. 29. Position 1. (From Dr. Erlanger's drawing.)

Fig. 30 shows the position "Out." Here the inflator is cut off, the tube (F), which leads to the whole manometer system, opens to the air through a bore in the bottom of the stop-cock, and T S again communicates with the exterior through E. In this position the pressure on the arm is immediately removed, but the tambour is protected from the violent change.

Fig. 31 shows position 1, which is used for the slow release of pressure in making measurements. The pressure space (P S) inside B, communicates with the fine capillary 1, in the bottom of the plug (C). The air escapes gradually through this, and the bulb B becomes smaller. The tambour head and lever sink, until the pressure in T S becomes so much less than the atmospheric pressure, that air begins to enter through the pinhole (O). The lever then ceases falling, and may even rise as the pressure slowly becomes equalized. The lever continues writing throughout all this procedure, reproducing the pulse-pressure changes of B in undiminished magnitude. Position 2 is the same, except that the capillary is a little larger, and the fall in pressure more rapid.

METHOD OF USE.—The armlet and inflator are attached to the tubes so designated in Fig. 27. The clockwork is wound. The drum is removed, covered with the glazed paper provided for the purpose, and lightly smoked. This is done by revolving it rapidly in the sooty flame of a fish-tail gas-burner, or lamp, or of burning camphor. The smoked cylinder is then set in place, and the instrument is ready for use.

Turn the stop-cock to "Out," and adjust the armlet snugly. Then rotate the stop-cock to "In," and raise the pressure rapidly by squeezing the inflator. The systolic pressure may be estimated in the usual way, by palpating the pulse, as with the Riva-Rocci type of instrument; or Erlanger's graphic method may be adopted (see pages 78 and 64). Not all cases lend themselves to this, in my experience, and the palpation of the return of the pulse may be necessary. For the other method, raise the pressure well (about 50 mm.) above the systolic pressure, then turn the stop-cock to an intermediate point for a few seconds. The pressure will fall slightly, owing to adjustment of the cuff, etc. The lever will show small oscillations. Bring the lever to bear on the drum, by turning the pinion-screw, and set the drum in motion. Now turn the stop-cock to 1 or 2, preferably the latter, after one has become expert. The lever will at first be depressed, then rise slowly, and the tracing will gradually increase in amplitude. The tracing and the manometer must both be closely watched, and the moment the pressure falls below the systolic pressure in the artery, the tracing will show an abrupt increase in size, usually with a depression of the lever (see Fig. 20). The manometer must be read off at this point, which gives the systolic pressure. This abrupt change is clearer with the more rapid fall of pressure through capillary 2, but it requires more practice to read the manometer at the exact moment. To determine the diastolic pressure, allow the air to continue escaping slowly until the pulsations show a distinct decrease in amplitude. The last point at which they are of maximum height is the diastolic pressure (see page 69).

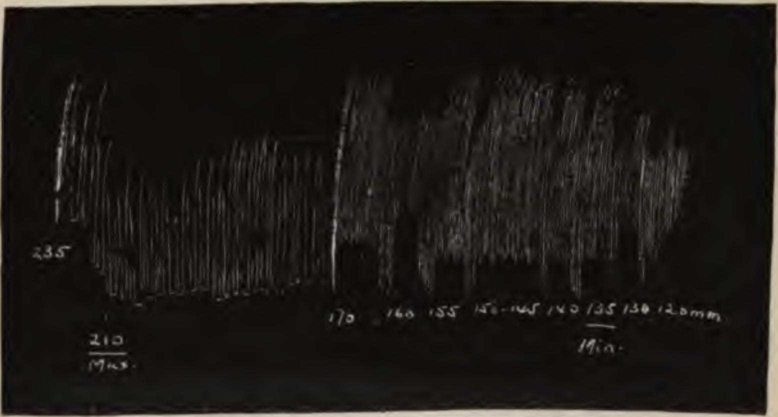


FIG. 32.—TRACING FROM A HIGH TENSION PULSE BY ERLANGER'S SPHYGMOMANOMETER.

At 210 mm. the return of the pulse indicated systolic pressure, though the trace does not show the abrupt increase clearly.
At 135 mm. is the lowest limit of maximal pulsation; diastolic pressure (marked Min.).

During the fall in pressure, a decrease in amplitude often occurs shortly after the systolic pressure has been reached, with subsequent increase. Fig. 32 shows these two maxima. The lower is unquestionably at the diastolic pressure. Erlanger considers the first decrease due to the rapid re-entrance of blood to the arm, the later increase coming when the vessels have been fully distended. This method gives a close approximation of diastolic pressure, but the following is more accurate.

Raise the pressure a little above the approximate diastolic, and, by manipulating the stop-cock, allow it to fall by steps of 5 mm., with the drum at slow speed. Take a record of ten or twenty pulses at each point, and the lowest pressure at which maximum amplitude of pulsation is obtained will be very evident, as in Fig. 32.

ADVANTAGES.—This is unquestionably the most accurate sphygmomanometer obtainable. It is also the best from the standpoint of workmanship. For the elucidation of physiological problems relating to human blood-pressure it is the ideal instrument.

DEFECTS.—The only fault to be found with the apparatus is from the side of clinical availability. It is bulky, and impossible to use in private practice, save in one's office. There, and in the hospital, I have employed it with much satisfaction. It requires considerable practice to work it easily, and to keep it in good order. Smoking the cylinder, and some of the other details, consume more time than can always be spared in practice, but the actual measurements take no longer than with any other instrument.

MAKERS.—The Southern Specialty Company, 2043 Hollins Street, Baltimore, Md.

COST.—$37.50 complete.

6. COMPARISON OF RESULTS OBTAINED WITH THE VARIOUS SPHYGMOMANOMETERS

A uniform method of blood-pressure measurement is much to be desired, and will certainly come with the general adoption of the 12 cm. armlet. In the meantime, reports of blood-pressure observations by various methods continue to be published. The difference in the readings is sometimes striking, particularly when figures from the Hill and Barnard or Mosso instruments are compared with those of systolic pressure determinations. The usual readings from Hill and Barnard's sphygmometer are neither diastolic nor systolic pressure, and cannot be compared with anything, so far as absolute values are concerned.

Of the forms giving systolic pressure, all those using the narrow armlet afford higher readings. For ordinary arms and normal tensions, this overestimation amounts to 10 to 25 mm. With marked hypertension and a large arm, I have found a difference of 60 mm. In children a 5 cm. armlet is sufficient. This means that one should know both circumference of arm and tension, in order to correlate the figures, rather a difficult condition to fulfil.

Comparison of Gärtner's tonometer with Riva-Rocci's sphygmomanometer, by Hayaski,[1] showed a lower estimation with the tonometer. The usual discrepancy was 20 mm.; the greatest 30 mm.; the least 16 mm. In normal cases Gärtner's tonometer would more nearly equal the readings with a wide cuff, but with higher tension the tonometer also tends to overestimate.

7. BLOOD-PRESSURE RECORDS

The value of graphic records is sufficiently obvious to-day to need no argument. We should know little of the characteristic temperature curve of typhoid fever, if our idea of it were obtained by reading a long column of figures. The course of the blood-pressure is equally easy to chart, and the curve thus obtained tells at a glance what long study of the numerical readings would probably fail to convey. In acute disease, or during operation, systolic pressure and pulse-rate should be charted at regular intervals. In more chronic cases a morning and evening record will answer; but, in cases of Bright's where uræmia is feared, frequent observations are quite as essential as the temperature record in a case of fever. Blood-pressure charts were first introduced by Cushing in his operative work. One of the most helpful additions to them is Cook and Briggs's use of a red line for the pressure, a black one for the pulse. The spacing in their charts, however, never seemed easy to follow. I have searched for a suitable ruling among the engineers' papers, but have failed to find it. In consequence I have worked out a chart for my own use. Instead of adopting arbitrary ruling, which may modify the curve, I have made 0.5 mm. = 1 mm., the scale of the U-tube manometer. In consequence, the height of the curve above the zero line represents the absolute pressure, on the same scale as a tracing from a recording manometer in the physiological laboratory. Each 50 mm. is a double line, and the 100 and 150 mm. lines are treble thickness. Between these, the normal systolic blood-pressure will usually be found. A single normal line, such as Cook and Briggs adopt, does violence to the facts, for reasons which will appear in the next chapter. For a similar reason I have been unwilling to couple any particular pulse-rate and tension. In my chart there is but one set of figures, so that the absolute pulse-rate can never be confused with the sphygmomanometer reading. To assist the eye, the figures are at both ends of the chart. The vertical rulings are 5 and 10 mm., so arranged that either twice a day, or every four, or every three hours, records may be kept equally well. For convenience in the operating room these charts are padded in blocks of fifty, and a double-ended pencil is used, marking the pressure in red, the pulse in blue. Where diastolic pressure is kept, a dotted red line indicates it, and a vertical dotted red line, connecting systolic and diastolic, represents the pulse-pressure. The respiration may also be recorded by a dotted black or blue line, an advantage in some cases.

---

[1] Hayaski, T. Vergleichende Blutdruckmessungen an Gesunden und Kranken mit den Apparaten von Gärtner, Riva-Rocci und Frey. Inaug. Dissert., Erlangen.. 1901, December.

One of these charts in full size, from a case of lobar pneumonia at City Hospital, is shown in Fig. 33. They are made for me by Mr. George W. Buskirk, Thirty-fourth Street and Sixth Avenue, New York. Having met so much difficulty in finding a convenient ruled paper for blood-pressure records, I have asked Mr. Buskirk to keep some of these charts on hand, in case they should be desired by others.

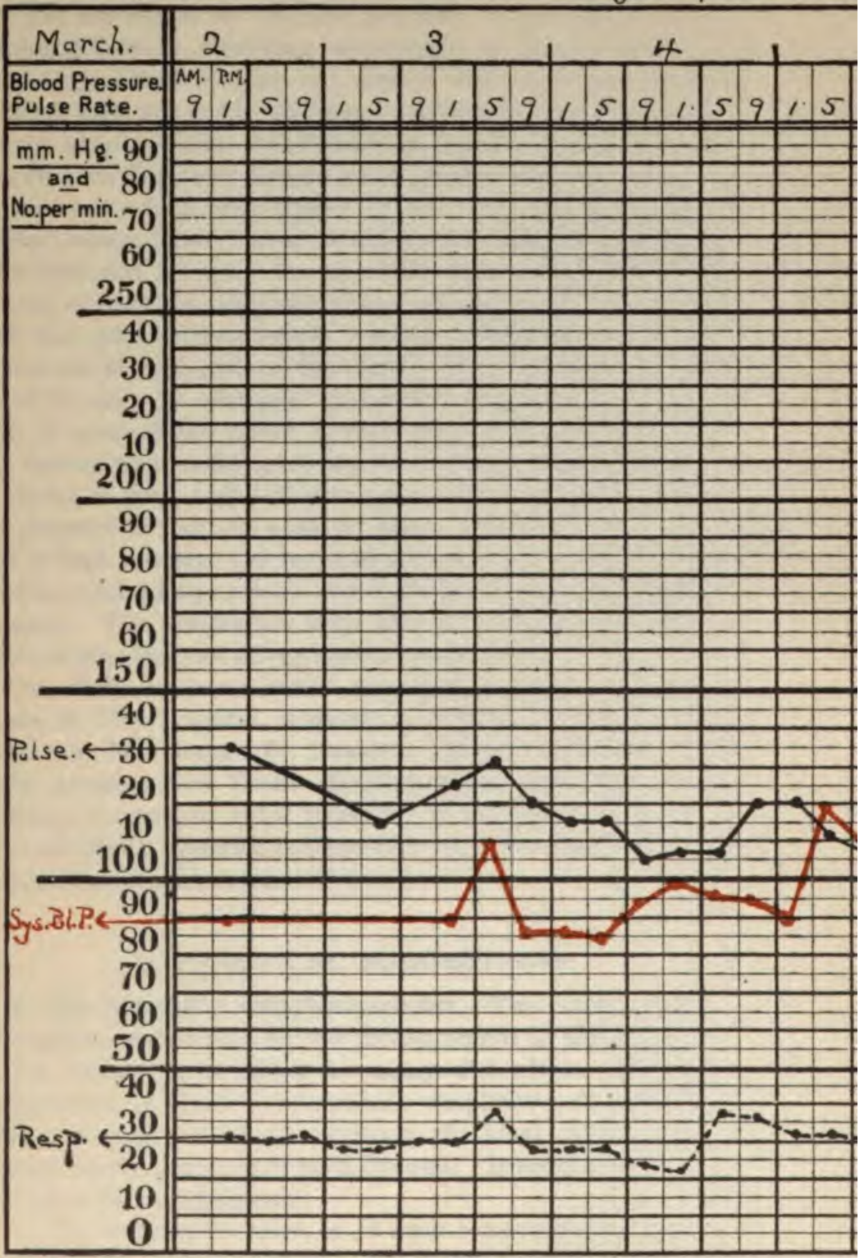
8. PRACTICAL SUGGESTIONS

A. The choice of a sphygmomanometer.—The construction of an accurate instrument for the measurement of blood-pressure at the bedside is attended by many difficulties. The most troublesome of these is to combine simplicity and portability with exactness. As a consequence, the ideal clinical sphygmomanometer has not yet been devised. It must comply with the following requirements:

1. The manometer must be of such construction as to give permanently exact readings. No metal manometer yet in-

Blood-Pressure

Date. 1904. Name. F. T. City Hospital, Ward

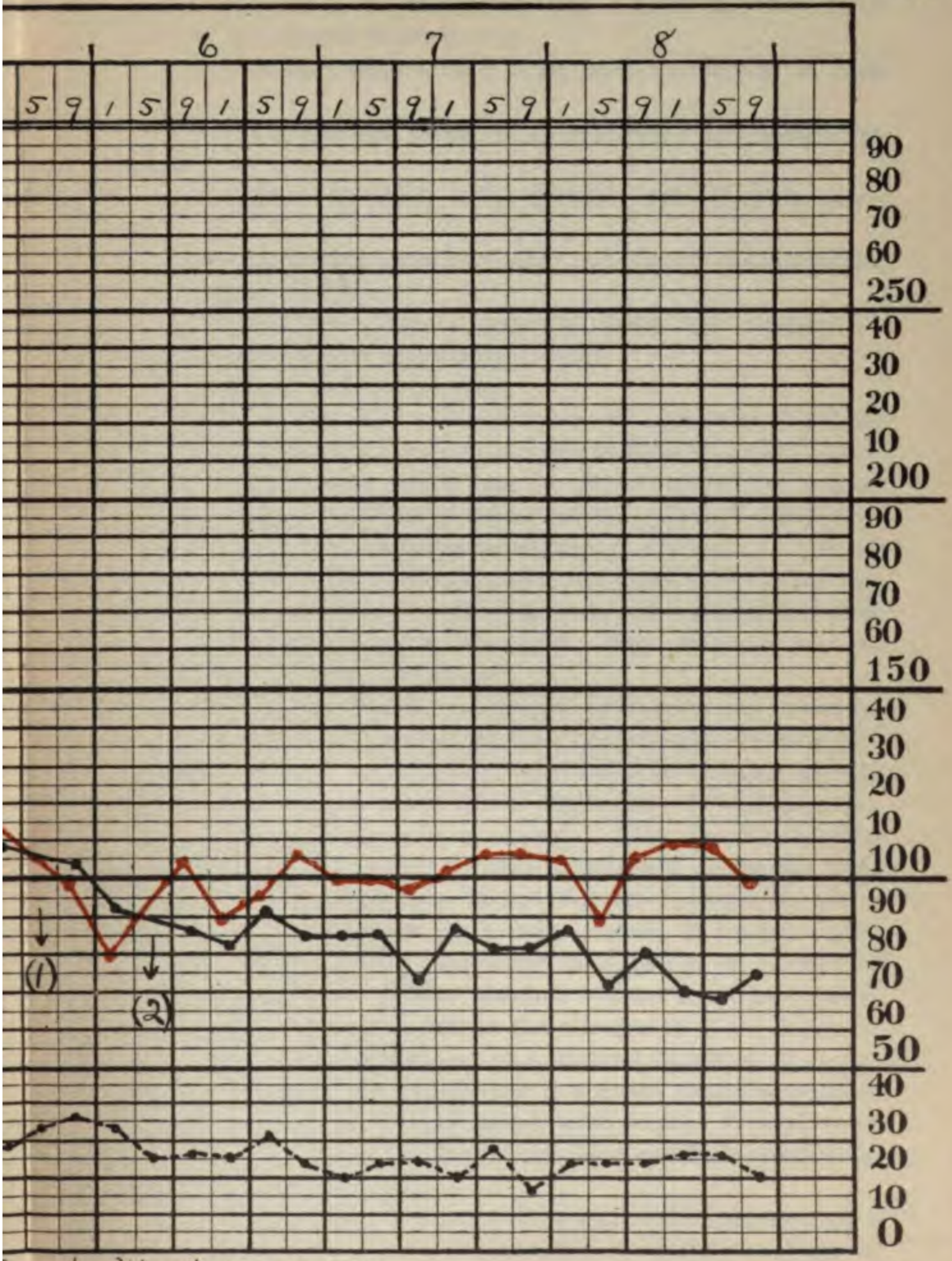


(1) Beginning of crisis. Temp. 103.2°. Until this point temp. h

(2) End of crisis. Temp. 99°. Lowest pressure during the cri

d Pulse Chart.

ale: Age. 23. Lobar Pneumonia. Recovered.



been steadily at or near 103°.

. Convalescence uninterrupted. Pressure never below pulse again.

vented remains accurate, hence this means the use of a properly graduated mercury one.

2. The compressing armlet must have a width of at least 12 cm.

3. The connections must be of practically non-distensible tubing.

4. It must measure both systolic and diastolic pressures.

5. Its application must be simple and require not more than two or three minutes.

6. It must be at once substantial, light, and compact, so that it may be easily and safely carried.

7. It should not be too costly.

When we test the existing forms of apparatus by these standards, we find none which are not lacking in some respect. The only instrument which can be recommended for experiments, which demand exact measurements of small differences in both systolic and diastolic pressures, is Erlanger's. Unfortunately it is bulky, requires some time and care for its use, and is rather costly. One must be somewhat acquainted with physiological technique to handle it. For office use, and for research, I consider it admirable.

For ordinary clinical, as opposed to careful experimental work, Stanton's sphygmomanometer and the author's are quite accurate enough. The estimation of diastolic pressure is only necessary in certain questions of diagnosis. For routine use, systolic pressure alone is necessary. This can be obtained with substantial accuracy by any instrument using the wide cuff. The only apparatus, except the two mentioned, which can be carried about on one's daily rounds, is Cook's. This cannot be relied on for diagnostic information on account of its narrow armlet. Where absolute values are unnecessary, and it is only desired to follow the changes in blood-pressure, in an attack of acute disease, or a surgical operation, it is perfectly adequate. The small cost is a great recommendation. Gärtner's tonometer, though much used to-day, I think must give place to methods employing the more satisfactory criterion of the return of the pulse.

B. Minor details.—To fill a manometer with mercury, use a medicine dropper. If the tube-calibre is small, the drops of mercury may fail to go down. A fine wire run down the tube will send the mercury to the bottom at once.

Never make a reading with a broken mercury column. Either shake it together gently, or blow it up and down with the inflator, until the air-bubble is forced out.

If your sphygmomanometer will not hold the pressure, examine all the joints for the leak. If not found, and the armlet is air-tight, there is probably a pinhole somewhere in the tubing. If it is not possible to get pulsation enough in the sphygmomanometer to estimate diastolic pressure, readjust the armlet more snugly before abandoning the attempt to measure it.

Never attempt determinations where there is œdema, much muscular twitching, or rigidity.

Practise using your sphygmomanometer on some normal people until you are sure of the method, and know a little of the variations of blood-pressure in health, before putting confidence in your results on patients. Following the effect of 1/100 gr. of nitroglycerin for half an hour, is an excellent way of acquiring familiarity with the instrument at different pressures. Making a diurnal chart is another good practical exercise.

In all readings for clinical purposes, have the patient absolutely at rest. In comparative readings on the same patient, the position must be the same, sitting or lying, with the arm at the level of the heart. In daily observations, the hour should be as nearly as possible the same.

PART III—CLINICAL

CHAPTER V

BLOOD-PRESSURE IN THE NORMAL MAN

1. **General considerations.**

2. **Blood-pressure in different individuals.**
   - A. Normal limits and influence of age, sex and occupation.
   - B. Influence of size and temperament.

3. **Blood-pressure variations in the same individual.**
   - A. The periodic variations.
   - B. The diurnal variations.
   - C. Influence of posture.
     - a. Influence of unaccustomed rest.
   - D. Influence of sleep.
   - E. Influence of meals.
   - F. Influence of alcohol and tobacco.
   - G. Influence of psychical states.
   - H. Influence of muscular exertion.
   - I. Influence of external temperature and baths.
   - J. Influence of atmospheric pressure.
   - K. Influence of menstruation.
   - L. Summary.
   - Literature.

CHAPTER V

BLOOD-PRESSURE IN THE NORMAL MAN

1. GENERAL CONSIDERATIONS

In the human being, as in the experimental animal, the blood-pressure at a given moment depends upon the interplay of four factors. The two predominant ones, cardiac energy and peripheral resistance, are to a high degree variable, but at the same time closely correlated through the vaso-motor and cardiac centres in the medulla oblongata. With such central control there is no a priori reason why mean aortic pressure might not be maintained at a practically constant level, as is the body temperature, and still leave ample scope for the adaptation of blood-supply which changing local conditions demand. Nevertheless, arterial pressure is no such constant. Its fluctuations greatly exceed those of the temperature under normal conditions, though the latter is less absolute than many persons suppose. Blood-pressure, both in health and disease, is more comparable to the rate of the heart or the frequency of respiration. Like them it has distinct normal upper and lower limits, between which it varies from person to person, and in the same individual from moment to moment. Like them also, it has wide and less defined limits, which it may approach under pathological conditions. To interpret rightly the blood-pressure in disease we must first study its course in health, under the influence of those changes in the individual and his environment which we class as physiological.

The data for this have been obtained for the most part with the Riva-Rocci type of apparatus and a 5 cm. armlet, or the Gärtner tonometer. In all cases the instrument, width of compressing surface, etc., will be given, using the abbreviations to be found in the front of the book. For a comparison of the results of the various types of sphygmomanometer see Chapter IV, section 6. In no case will figures derived from the v. Basch or similar form be used in the discussion of absolute values, but only where comparative readings in the same individual are involved, and here with considerable scrutiny. The collation of figures relating to blood-pressure will be most difficult until the 12 cm. armlet has been universally adopted. In the meantime it is imperative that no results be reported without details as to method and width of cuff used, and circumference of arm. In every case systolic pressure is to be understood unless the contrary is specifically stated.

2. BLOOD-PRESSURE IN DIFFERENT INDIVIDUALS

A. Normal Limits and Influence of Age, Sex and Occupation.—In young adults there can be little question that 90 mm. (R. R.), 80 mm. (G), represents the lowest systolic blood-pressure that can be considered normal. The upper boundary is more dubious, because the errors involved in the method, especially those inherent in the narrow armlet, are on the side of overestimation, and it is hard to eliminate the possibility of causes for pathological high tension. For practical purposes, the greatest height to which systolic pressure may rise under purely physiological conditions is of no interest, though it might satisfy a harmless curiosity. Avoiding all causes, psychical and physical, for an increased blood-pressure, 160 mm. (R. R. 5 cm. and G.) is the usually accepted upper limit. With the 12 cm. cuff I think 145 mm. will prove nearer the absolute value. In more than 200 determinations, I have only once or twice seen it exceeded by a few mm., except where cause for hypertension existed. In the great majority of young males 100 to 130 mm. will be found. Females tend to have a systolic pressure lower by perhaps 10 mm., but with the same general limits. The larger differences sometimes found have probably been due to the smaller female arm.

In children under two years the pressures are undoubtedly lower. According to Cook and Briggs, 75 to 90 mm. are the usual figures. In older children the values are the same as the lower ones found for adults, and the limits of variation are smaller. These data have much more absolute value than our present ones for adults, for two reasons. In children there is little likelihood of errors due to the size of the arm; and, in the second place, beginning cardio-vascular change can be practically excluded. When we come to the other extreme of life, both these causes of abnormally high readings come into prominence. Obesity is far more common after middle age, and changes in the small vessels may give no other evidence of their onset than an increased arterial pressure. For these reasons I think we must be very cautious in assuming that the higher pressure so commonly found in elderly people is really a normal phenomenon; certainly one finds moderate figures even in the aged. In the routine examination of many hundred cases during the last three years, I have never seen a pressure above 180 mm. (R. R. 5 cm.) in a normal person, and seldom one above 160 mm. Gumprecht puts 200 mm. as the upper limit in old people; Hensen, 170 mm. These figures, of course, hold good only during physical and mental rest. With the broad cuff they should be 20 to 30 mm. lower. If we ask the usual values, not the possible ones, we find that, while in childhood the blood-pressure is most frequently at its lower normal levels, after fifty the average is considerably above that of early maturity. One expects 140 to 160 mm. (R. R. 5 cm.), 130 to 145 mm. (12 cm.).

The cause of this rise of blood-pressure in later life has been variously explained by narrowing of the arterioles or of the capillaries, increased viscosity of the blood, and so on. At least we may feel justified in assuming that it is due to increasing peripheral resistance, whatever be the more remote causes. It manifests itself also in a more sudden and marked reaction than follows in the young, when influences which increase pressure come into play. This we shall see especially under exertion.

The influence of occupation on blood-pressure has been little studied, but Gumprecht found, in men accustomed to hard manual labor, figures similar to those which obtain in old age. Here again the greater muscular development and the frequency of vascular disease, in the laboring classes, make me accept his results conditionally only.

For the diastolic pressure few figures can be found in the literature. On the basis of several hundred readings in about two hundred cases, besides a large number of approximations by my former method, I should place the normal at 25 to 40 mm. below the systolic pressure in the same person. This holds good only during rest, for posture, exertion, etc., affect the two pressures unequally. On the same basis, not as yet a perfectly adequate one, the normal limits of diastolic pressure would be between 65 and 110 mm. The diastolic pressure

tends to vary less than the systolic under most of the conditions which increase tension, especially the pathological ones. It is therefore a less satisfactory guide when taken by itself. Taken in connection with systolic pressure it is most valuable. Erlanger calls the difference between the two the pulse-pressure. He and Hooker have shown that, under perfectly normal conditions, the product of pulse-pressure by pulse-rate tends to remain constant. This is probably a more accurate statement than Marey's, that blood-pressure and pulse have an inverse relation to one another.

Riva-Rocci, 5 cm. Armlet.

		Highest.	Lowest.	Average.
Cook and Briggs:				
Children up to two years.	75– 90 mm.			
Children after two years..	90–110 "			
Young adult males, about	130 "			
Women	10– 15 " lower.			
Gumprecht:				
Children	90–110 "			
Women	120 "			
Men	140 "			
Laborers and aged men...	160–200 "			
Hayaski:				
108 healthy children (9–14 years)		141 mm.	102 mm.	118 mm.
58 men below sixty (excluding some cases of disease not excluded by him)		180 "	96 "	132 "
Women		170 "	100 "	128 "
Hensen:				
25 children		150 " (next 135 ")	80 "	116 "
25 healthy laborers (17–30 years)		158 "	105 "	137 "
30 healthy women		160 "	105 "	132 "
Thayer:				
276 healthy individuals arranged by decades.				
37 cases 1–10 years				104.6 "
87 cases 10–20 "				128.7 "
89 cases 20–30 "				136.9 "
37 cases 30–40 "				140.8 "
20 cases 40–50 "				142.2 "
5 cases 50–60 "				154.8 "
1 case 60–70 "				180 "

GÄRTNER'S TONOMETER, 1.5 CM. RING.

		Highest.	Lowest.	Average.
Doleschal:				
100 persons				120 mm.
Gärtner	105–130 mm.			
Grebner und Grünbaum	110–125 "			
Hayaski:				
108 healthy children		120 mm.	85 mm.	97 "
58 men below sixty (see above)		160 "	80 "	111 "
Women		145 "	80 "	106 "
Jackson:				
Young men, upper limit	140–150 "			
Old people, upper limit	175 "			
Jellinek:				
532 healthy soldiers		185 "	80 "	
Great majority between	100–160 "			
Kapsamer				120–130 "
Schüle:				
Normal limits	80–130 "			
One-half his cases	100–110 "			
Shaw:				
45 children				90–100 "
Weiss:				
Men, normal limits	90–120 "			
Women, normal limits	80–100 "			

HILL AND BARNARD.

		Highest.	Lowest.	Average.
Carter:				
Males				116 mm.
Females				113 "
Hill			(Sitting)	120–125 "
Jarotzny:				
Adults			(Sitting)	110–130 "

POTAIN.

Potain, with his instruments, considers 170 mm. the average pressure for adults; 140–210 mm. the normal limits.

v. BASCH.

v. Basch makes 110–140 mm. the normal variation. 150 mm. he considers hypertension.

From the systolic and diastolic pressure the mean pressure can be calculated with considerable accuracy by taking their arithmetical mean, as Howell and Brush demonstrated (see page 34). Since the form of the pulse-curve is always a triangle, with the apex upwards, I feel convinced that the absolute mean pressure, which could only be calculated with some difficulty, must be nearer the diastolic than the systolic level. However, the average of the two is quite near enough for practical purposes.

The most trustworthy figures for normal human blood-pressure are given on pages 110 and 111.

B. Influence of Size and Temperament.—Greater size and body-weight have been said by many writers to be attended by higher blood-pressure. Until this connection has been demonstrated with the 12 cm. armlet, it seems quite probable that such persons have given erroneously high values.

Temperament seems to have a real influence, from the fact that the more excitable and neurotic individuals show a greater rise in pressure than the phlegmatic, from the same psychical cause. The daily pressure-curve of such persons will have a wider range, and single measurements of blood-pressure will more often be unduly influenced by excitement or apprehension. With this nervous tendency one must reckon in the use of the sphygmomanometer for diagnostic purposes.

3. BLOOD-PRESSURE VARIATIONS IN THE SAME INDIVIDUAL

A. The Periodic Variations.—These are evident in the human being as in the animal. The respiratory and the Traube-Hering waves, and the other less rhythmical but apparently spontaneous fluctuations in mean blood-pressure (see page 35), must be in mind during every clinical experiment. The respiratory waves are usually negligible during quiet breathing, though I have been able to measure a 10 mm. rise in pressure during expiration, in some people. In experiments on the effect of exertion, when measurements are made during deep labored breathing, they will be very evident. Especially must it be remembered, in comparing the effect of exercise and similar procedures on the blood-pressure, when investigated with different instruments, that neither the respiratory nor Traube-Hering curves affect systolic and diastolic pressure equally. In both cases, with the rise in mean pressure there is an increase in the size of the pulse-wave. This can be seen beautifully in the curves reproduced by Mosso (Fig. 34). It predicates a greater effect on systolic pressure than on diastolic, and a consequent divergence in results between the two types of clinical instruments. Exact figures for the possible pressure variations due to these causes are hard to give, but their extent in animals, combined with my observations on the changes noted in patients from moment to moment, lead me to place 30 mm. Hg. as the probable maximum rise which may be attributed to them. One sees these larger fluctuations mainly in patients with hypertension. Ordinarily, 5 to 10 mm. would be a liberal estimate.

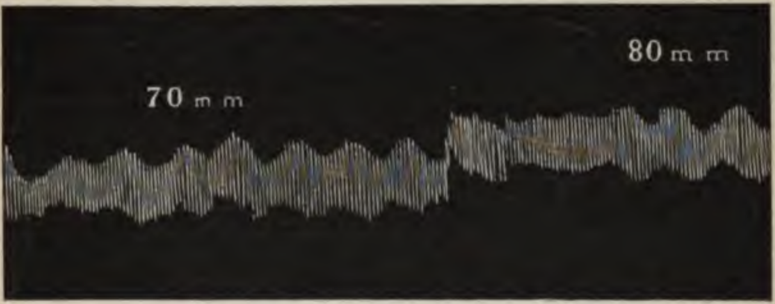


FIG. 34.—PERIODIC FLUCTUATIONS IN THE TRACING OF BLOOD-PRESSURE TAKEN FROM DR. COLOMBO (WHILE AT REST). (Mosso's sphygm.)

(From Mosso, Arch. Ital. de Biol., 1895, vol. xxiii, p. 192, Fig. 7.)

A due appreciation of these facts makes some of the discrepancies in the findings of different observers intelligible. I do not quite agree with Hensen that, in blood-pressure experiments, no conclusions must be drawn from changes of 10 or 20 mm. Where a chart of the pressure, at intervals of a few minutes, is kept for a considerable period before and after the procedure to be investigated, small differences can be accorded distinct value. This is especially true if each figure represents the average of two or three successive readings.

B. The Diurnal Variations.—A record of blood-pressure taken at frequent intervals throughout the twenty-four hours shows deviations from the average level more striking than those we have already considered, and of too great extent and too long duration to be explained by them. Such a chart is shown in Fig. 35. Evidently it is a composite record of the effect of various physical and mental states on the blood-pressure. It would differ from individual to individual, and somewhat from day to day. The total diurnal variation from all causes probably seldom exceeds 60 mm. Hg. Such extreme fluctuations have not come within my experience. Goldwater, and Weiss (G.) picture them. Besides the more obvious causes for fluctuation, which we shall consider separately, there are unquestionable periodic rises and falls in blood-pressure at different hours of the day, entirely unassociated with changes in external condition. Colombo has studied these especially, and gives a table and chart of diastolic pressures, taken with Mosso's

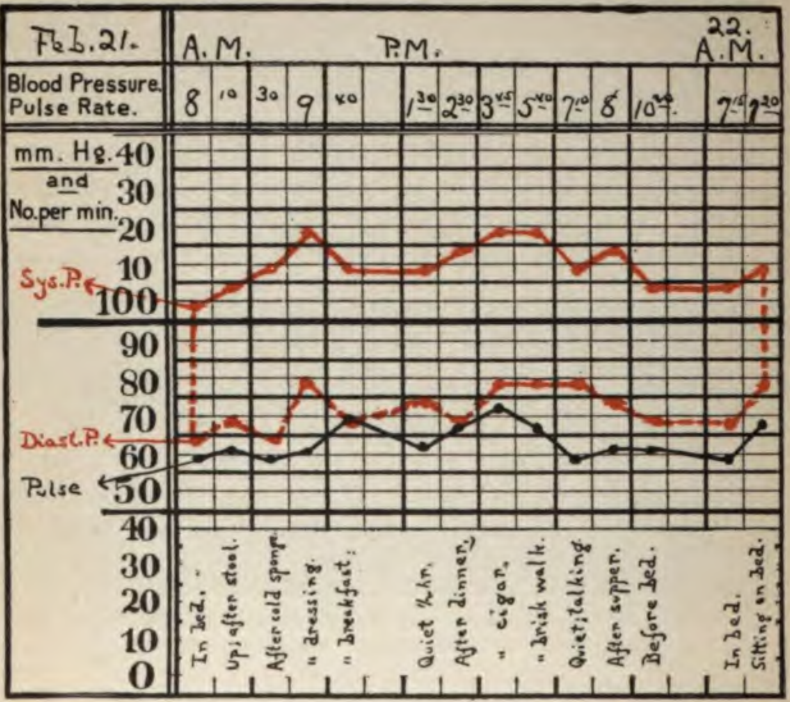


FIG. 35.—DIURNAL BLOOD-PRESSURE CHART OF A HEALTHY MAN.
(Author's sphygm., 12 cm.)

instrument at half-hour intervals throughout the twenty-four, which shows fluctuation between 65 and 100 mm. (Fig. 36).

The supposed periodic afternoon rise in pressure of certain observers (Zadek, Hensen, Jellinek) is not constant, and can probably be attributed to psychical activity, as Goldwater suggests.

We shall now take up the chief influences which affect blood-pressure in more detail.

C. Influence of Posture.—This must not be confused with the effect of gravity (see page 59), which should be eliminated by making all determinations at the level of the heart. The results of concomitant physical and mental effort must also be allowed to pass away before judging the influence of bodily posture.

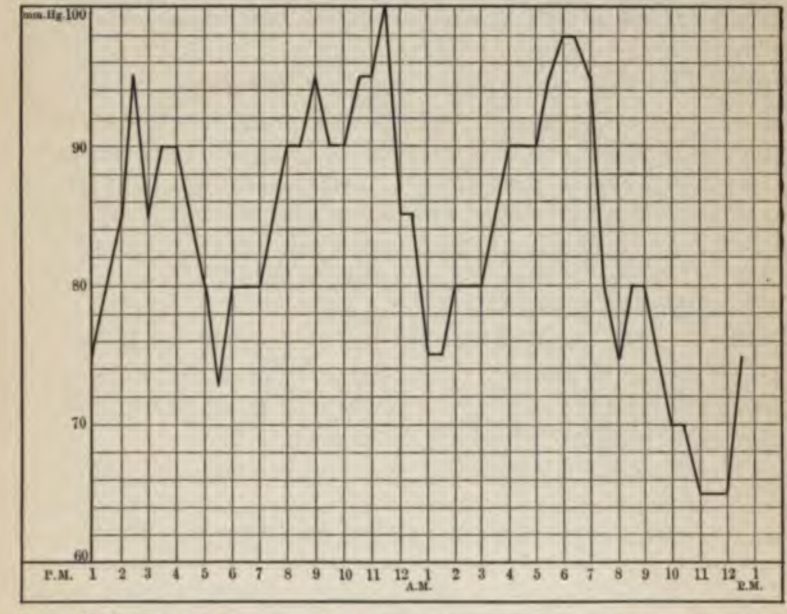


FIG. 36.—DIURNAL VARIATIONS IN DIASTOLIC PRESSURE, THE INFLUENCE OF MEALS BEING EXCLUDED. (Mosso's sphygm.)

The wide spacing of this chart exaggerates the changes depicted. (From Colombo, Arch. Ital de Biol., 1899, vol. xxxi, p. 368.)

With such possibilities of error, it is not surprising that difference of opinion exists, especially since the rise of pressure due to change of position is so small as to fall well within the limits of spontaneous periodic variation. The weight of evidence seems to point toward the recumbent posture as conducive to the lowest pressure, standing and sitting being alike in raising both systolic and diastolic pressures 5 to 10 mm.

Ekgren in 300 cases found the pressure averaged 5 mm. (R. R.) lower in the horizontal position. Cook and Briggs give 5 to 10 mm. (R. R.) as the usual difference: Gärtner, and Goldwater 15 mm. (G): Hill, about 10 mm. (H. & B.): Brush and Fayerweather, 10 mm. (Mosso). Hensen, however, thinks pressure lowest when standing, and quotes Friedmann and Schapiro on this side, as assuming a fall of 10 to 15 mm. on standing. v. Recklinghausen came to the same conclusion, but apparently from a single experiment only, and in that the pressure when lying without a pillow was 15 mm. lower than lying or sitting, 6 mm. below that on standing. Karrenstein's 60 determinations with the tonometer showed an average fall of 6.7 per cent. on lying, 3.7 per cent. on standing, as compared with sitting. I have usually found a slight fall on lying down, in normal cases.

The most convincing evidence on this subject is found in the recent work of Erlanger and Hooker. They found the pressures about the same sitting or lying, but on standing, diastolic pressure rose much more than systolic, the pulse-pressure variation being diminished. Comparison with the v. Kries tachygraph, which showed the greatest acceleration of blood-flow per heart-beat in the recumbent posture, confirmed their observations.

Evidently the postural variations of systolic pressure are insignificant. The main deduction to be made from the somewhat conflicting testimony is, to make all comparative tests in the same position, preferably the horizontal one.

a. Influence of Unaccustomed Rest.—Under this heading we must allude to Gumprecht's observations on the effect of unaccustomed rest on blood-pressure. He found that laboring men with pressures between 160 to 200 mm. (R. R. 5 cm.) on entrance to the hospital, showed a considerable fall after a few days in bed. Hensen denies any effect of this kind. He calls attention to the higher reading usual on the first day in a hospital, which he considers due to excitement.

D. Influence of Sleep.—Unquestionably pressure is lowest during the first hours of natural sleep, rising slowly toward the time of waking. Whether the fall in pressure stands in any causal relation to the sleep, or whether both must be considered effects of a more remote cause, cannot be stated. The prompt hypnotic action of erythrol tetranitrate, a drug causing vaso-dilatation only, in the cases of insomnia with high tension studied by Bruce, is certainly suggestive. The effect of posture, and the absence of all psychical influences which raise pressure, seem adequate to explain the normal fall, but not the subsequent gradual rise.

Brush and Fayerweather found that diastolic pressure fell during the first few hours of normal sleep, which corresponds with the minimum pressure in Colombo's chart, between 11 and 12 P. M. Later the pressure rose gradually, but not uniformly, the curve showing long waves. On awaking, the pressure was somewhat higher than before sleep. Hill, however, did not believe that blood-pressure was any lower during sleep than when resting in the recumbent position. Kornfeld, in his studies on trional, found that the fall in pressure after taking the drug was much greater if the hypnotic effect were obtained, than if not. He attributes the whole action to a diminished irritability of the nervous system.

E. Influence of Meals.—The effect of the ingestion and digestion of food on arterial pressure is another disputed point, so much so that it is wiser to suspend judgment on it until more complete data are available. Here again many other factors may enter in and be the cause of wide individual variation.

Colombo's charts show a constant fall in diastolic pressure up to 20 mm. (Mosso), after meals, absolutely independent of the time of day at which they were eaten. No other work on the subject has been so carefully performed. Weiss found a similar fall. Gumprecht, on the other hand, frequently found a slight rise in systolic pressure, up to 20 mm. (R. R.), after the hearty meal of the day. With the Gärtner tonometer Jellinek found in 14 out of 20 soldiers a rise, no change in 4, and a fall in 2. Karrenstein saw a rise of 10 to 20 mm. commonly after the principal meal, and never failed to get an elevation of 10 to 25 mm. if much fluid (2 litres of beer) were drunk. Sommerfeld considered the midday meal the cause of a rise, and Hayaski never missed it. A most interesting observation of Cook's was the regular rise of 5 to 10 mm. in systolic pressure in infants, following nursing from the bottle.

In my chart (Fig. 35) a rise of 5 mm. in systolic, and a fall of 5 mm. in diastolic pressure, followed both dinner and supper.

Those who find a lower pressure during digestion assume with apparent justice that it is due to vaso-dilatation in the splanchnic area. It may be that some individuals have accompanying this an amount of superficial vaso-constriction which overcompensates (see page 24). Thus they would evince an actually higher pressure. I am inclined to believe, however, that studies of the pulse-pressure variation will show that systolic and diastolic pressure are unequally affected by the taking of food, and that the discordant observations quoted may thus be harmonized.

F. Influence of Alcohol and Tobacco.—The enjoyment of some form of alcoholic beverage, and of tobacco, so commonly accompanies the normal man's meal, that the influence of these drugs on blood-pressure may be best discussed here.

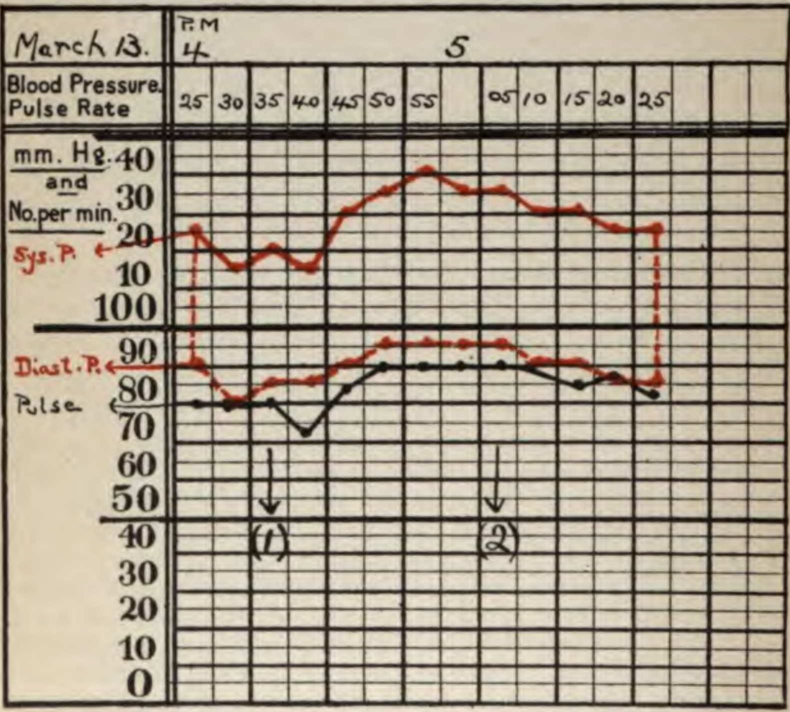


FIG. 37.—RISE IN BLOOD-PRESSURE PRODUCED BY TOBACCO.
(Erlanger's sphygm., 12 cm.)

Chart obtained from a healthy young man, sitting quietly, who smoked a strong cigar between (1) and (2).

Alcohol, contrary to the usual belief expressed in the language of daily life, is not a stimulant, at least so far as the circulation is concerned. The pharmacologists agree that it produces no rise in blood-pressure or in cardiac energy when injected directly into a vein. On the contrary, if a considerable dose be given, arterial pressure falls from weakening of both vaso-constrictor centres and heart (see Cushney). In man there seems no good reason to doubt that its effect is the same. Swientochowski, Schüle, and Wren and Oertel have, in fact, demonstrated that no elevation of systolic pressure follows its administration. Observations on the pulse-pressures are lacking.

Tobacco, on the other hand, or its alkaloid nicotin, have a powerful action on the circulation. Nicotin, in less than overwhelming dose, produces an immense augmentation of blood-pressure in animals, due to stimulation of both central and peripheral vaso-constrictor mechanisms (Cushney). Cook and Briggs have called attention to the temporary elevation of arterial tension during smoking. They found it most marked when a strong cigar or old pipe is used, and continuing an hour or more after the smoke is ended. This latter statement I have been unable to confirm (see Fig. 37).

G. Influence of Psychical States.—When we come to study the effect of different psychical states on blood-pressure, we fortunately meet with more unanimity of opinion. Excitement seems indubitably the most powerful cause of increased pressure in the normal man. A heated argument may raise the systolic pressure in a short time as much as 40 mm., as is shown in Goldwater's diurnal chart (G). The accompanying chart (Fig. 38) shows a moderate increase in systolic pressure and pulse-rate, after lecturing an hour to a large class, certainly a considerable mental effort, with some excitement. Diastolic pressure was much less affected. Especially striking is the rapid fall, to below the initial pressure in less than six minutes, after the cessation of the effort.

Goldwater, and Kapsamer (G), Gumprecht (R. R.), and Hill (H. & B.), have recorded equally pronounced changes, and every one who uses a sphygmomanometer may observe them. Anger is especially potent in its effect. Some attempt has been made to differentiate the effects of the separate forms of cerebral activity, perceptive, volitional, intellectual, etc. Kiesow made a number of experiments with Mosso's sphygmomanometer, in which he showed a distinct rise in diastolic pressure with the intellectual effort of a simple arithmetical calculation, with mild fright, with disagreeable taste perceptions, but not with odors. With sight impressions, even when the closest effort of attention was involved, he found no influence on the curve. Gumprecht also found the mere effort of attention usually without effect on systolic pressure (R. R.); sometimes a rise of 5 or 10 mm. was produced. One student, when he read the manometer himself, always showed 15 mm. higher pressure than when he did not. Even the slighter emotional conditions, such as gentle laughter, were enough to cause a considerable increase (16 mm. R. R.). Pain has a similar effect. The explanation of these marked influences must be sought in the well-known reflex vaso-constriction which follows stimulation of sensory nerves in general, and of the cerebral cortex (see page 21). One is tempted to think of the dependence of the brain circulation upon general blood-pressure to meet its local demands, since the brain vessels probably have little vaso-motor supply, as the possible cause of the increased aortic pressure which accompanies cerebral activity.

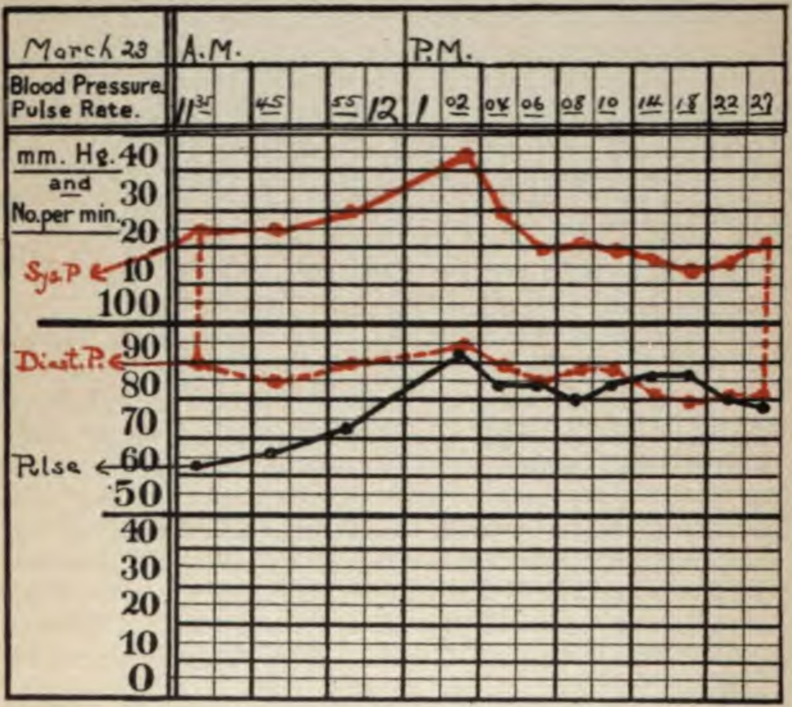


FIG. 38.—RISE IN BLOOD-PRESSURE PRODUCED BY MENTAL EFFORT. (Erlanger's sphygm., 12 cm.)

Chart obtained from Prof. ——, who lectured to a large class from twelve to one o'clock. Before and after lecture he sat quietly in his laboratory.

Note the coincident rise in pulse-rate; the greater rise in systolic than in diastolic pressure; the subsequent fall in pressure, with decreased pulse-pressure, while the rate remained rapid.

The meaning of these psychical influences for the clinician is obvious. In all determinations of blood-pressure they should be conspicuous by their absence, and the physician must sedulously guard his patient against undue interest in the procedure itself, or fear of it, and secure as complete mental relaxation as possible during the measurement.

H. Influence of Muscular Exertion.—That muscular work usually increases systolic blood-pressure in man has been known since the early investigations with the v. Basch sphygmomanometer. This, of course, tallies with the results of Tangl and Zunst in dogs (see page 39). Zadek, and Maximowitch and Rieder found the rise in pressure proportional to the work done, reaching as much as 50 mm. Hg. with young men undergoing severe exertion, falling rapidly after its cessation, and becoming again normal within twenty to thirty minutes. Their results have been verified by practically all recent observers. Among the latter, Jellinek, Grebner and Grünbaum, Kornfeld, Masing, and O. Moritz have investi-

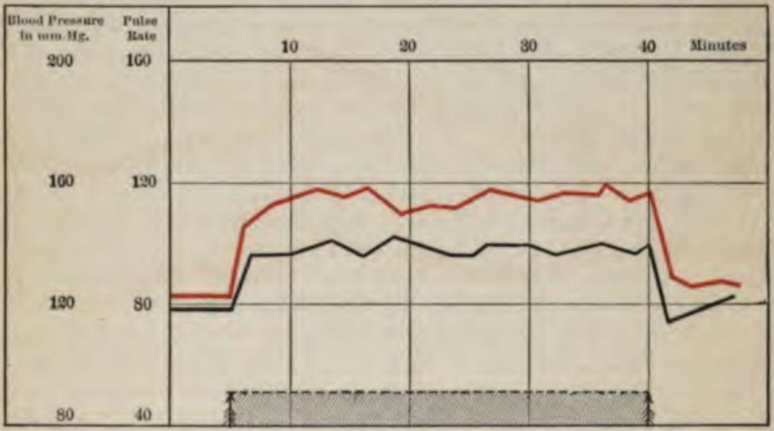


FIG. 39.—RISE IN BLOOD-PRESSURE PRODUCED BY MUSCULAR EXERTION.
(Riva-Rocci's sphygm., 5 cm.)

Chart obtained from a student of twenty-two, who performed, with both legs, 88 kilogrammeters of work a minute. At the close of the work, slight fatigue of the legs, and somewhat deepened respiration.

The hatched area below indicates the duration of the work.

Note the rapid rise in blood-pressure and pulse-rate, maintained throughout; and the equally rapid return to normal. (From Masing, Deutsch. Arch. f. klin. Med., 1902, vol. lxxiv, p. 283.)

gated this question particularly, and have brought to light certain facts which require more detailed consideration.

O. Moritz, Masing, and Kornfeld, all emphasize the psychical factor in muscular effort. This manifests itself unmistakably in their experiments. The greater the conscious effort the greater the rise in pressure, and vice versa; so that pressure seems proportional rather to the fatigue produced than to the actual work done. Work performed with a single leg raised pressure more than the same amount done by the two legs alternately, and the first performance more than subsequent ones (Masing). Kornfeld found that the attempt to walk in a straight line had more effect than the same distance without any special attention. Moritz goes so far as to state that the pressure elevation during bodily work is principally conditioned by psychical influences. These facts explain at once the effect of practice, as in athletic training for instance, which consists essentially in such repetition that coördinations become almost automatic reflexes, and the voluntary effort is reduced to a minimum. They also make intelligible the few discordant observations in which exercise did not raise pressure, such as Jellinek's on 106 soldiers, in whom marching raised pressure in 57, had no effect in 30, and was attended by a slight fall in 19 (G). Here, apparently, the effort was insufficient of itself to affect 49 of the men, and other factors determined the results found. The individual differences reported by Schüle, and Huber, may be similarly explained.

The relative effect of exertion on systolic and diastolic pressures has been investigated by Masing. By a method yielding approximate values only, he found that both systolic and diastolic pressure rise during exertion, but in different degree, the diastolic always less. This is in accord with the well-known increase in pulse-volume. One experiment of mine with the Erlanger apparatus (5 cm. armlet) showed the following in a healthy young man of twenty-six years.

	SYSTOLIC PRESSURE.	DIASTOLIC PRESSURE.
Sitting.........	135 mm. Hg.	100 mm. Hg.
Lying..........	125 mm. Hg.	95 mm. Hg.
After running up three flights of stairs. Sitting.	175 mm. Hg. = 40 mm. increase	120 mm. Hg. = 20 mm. increase

It follows then, that instruments measuring systolic pressure will overestimate, those giving diastolic will underestimate, the effect of exertion on mean aortic pressure. Here, as elsewhere, the determination of both pressures would be most valuable in throwing light on the actual vascular changes. The indications afforded by the increased pulse-volume, in the present instance, point clearly to increased energy of the heart as at least one factor in the rise of pressure, if not the only one. Under conditions of too excessive work in proportion to the individual's cardiac power, the blood-pressure, after its primary rise, may show a gradual fall, even below the normal. With this there are dyspnœa and other subjective symptoms, and, according to Schott, and Larrabee, acute dilatation of the heart. Here we border on the pathological effects seen in diseased hearts, which will be considered later. Masing has found similar results from moderate exertion in the aged, the primary rise of pressure being maintained for only a short time. In these older men there was also less acceleration of the heart than in the young. Here again is strong evidence that the rise during physical work is due to increased cardiac energy.

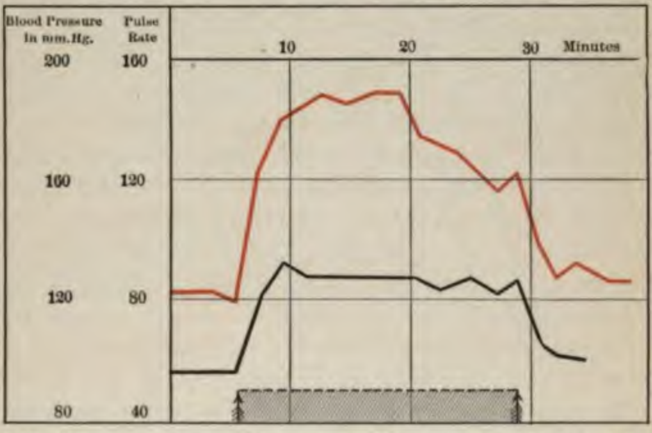


FIG. 40.—INFLUENCE OF MUSCULAR EXERTION ON BLOOD-PRESSURE IN THE AGED. (Riva-Rocci's sphygm., 5 cm.)

Chart obtained from a man of sixty-eight, with the same work as the subject of Fig. 39. During the work the pulse became quite irregular at times; after seventeen minutes, dyspnœa. At the close of the work, dyspnœa, palpitation, great fatigue of the legs.

Note the great rise in blood-pressure as compared with pulse-rate; its fall during the work; the more gradual return to normal at its close. (From Masing, loc. cit., p. 286.)

Karrenstein has just published observations on 74 soldiers undergoing rather protracted exertion; in one test, a march of about seventy minutes' duration over a considerable hill; in the other, three hours' exercise. The great bulk of his men showed a fall, most of those who did not having drunk considerable beer. He considers that in long-continued exertion a fall in pressure is common. Since he also noticed some increase in heart dulness in many cases, this fall seems analogous to that which occurs earlier in the aged and those with weak hearts. One is also tempted to postulate a depressor fall, as a protective reflex from the heart.

Finally it must be remarked, that the curves of pressure during exertion show irregular rises and falls, which are difficult of explanation (Grebner and Grünbaum, Masing). The most marked falls accompany profuse sweating.

We may summarize these facts as follows:

The rise in pressure during physical exertion is in general proportional directly to the amount of work done and to the voluntary effort required, the latter factor being the more important. The effect on systolic pressure is more marked, that on diastolic less marked than on mean pressure.

The rise is due to increased energy of the heart (both rate and pulse-volume), and, when the demand exceeds the reserve force of the heart, it will not be maintained. With equal work it is of much shorter duration in the aged than in the normal adult.

Prolonged exertion usually causes a fall in pressure.

I. Influence of External Temperature and Baths.—The effect of temperature changes upon the local circulation has long been well known and commonly used in practice. Cold produces local vaso-constriction, which, if sufficiently long continued, eventually gives way to a dilatation, due to paralysis of the vaso-motor mechanism. Besides this, when cold is applied for a short time and then removed, a reaction follows and the part becomes intensely hyperæmic, a sign of secondary active vaso-dilatation. Warmth, on the other hand, produces vaso-dilatation and local hyperæmia from the outset, probably of a passive nature; that is, from diminished vaso-constrictor tone.

If the local area thus subjected to changed temperature conditions be sufficiently large, a marked influence will be exerted on the distribution of the blood throughout the body, and, if there be no counterbalancing change elsewhere, on the general blood-pressure. It is just at this point that complications arise. We have seen (see page 21) that, not only do external irritations of any kind produce reflex vaso-motor effects in remote vascular areas, in that innervated by the splanchnic nerves especially; but also that the blood-vessels of the skin and those of the abdominal viscera frequently act in antagonism to one another. For this reason it is impossible to predict that the application of cold sufficient to constrict all the superficial arterioles will necessarily raise aortic pressure; for a reflex dilatation of the splanchnic vessels might more than compensate for the added resistance. The question must evidently be approached experimentally.

Here we at once meet the difficulty, that the necessary procedures may be attended by physical exertion and mental excitement, with their concomitant vascular effects. In almost all the work upon the subject these have not been carefully enough eliminated. In addition, much of the investigation has been with the inaccurate v. Basch instrument; as, for instance, Jakob's work, which I cannot place any reliance in. The Gärtner tonometer, and Mosso's sphygmomanometer also, must be fallacious for this purpose, because influenced by the local vaso-motor conditions induced in the fingers; hence Jellinek's, and Tschlenoff's results cannot be accepted. Edgecombe and Bain studied the question of baths and exercise with considerable care, but used Oliver's hæmodynamometer. Their results agree in the main with the experiments which follow, and add nothing to them. The most careful investigations I have been able to find are those of Bruck, and of Müller.

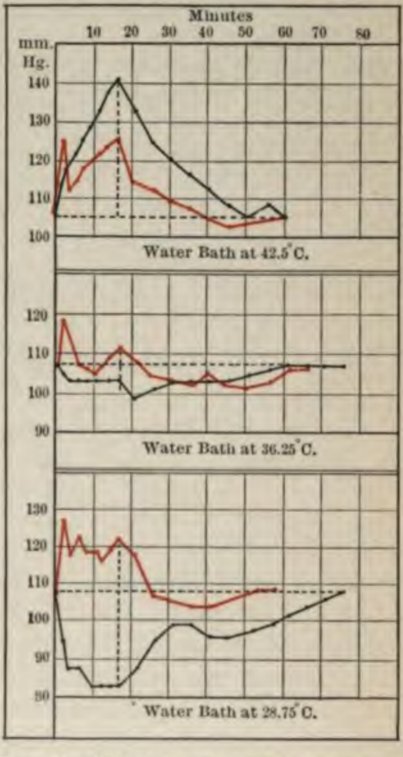


FIG. 41.—INFLUENCE OF WATER BATHS OF DIFFERENT TEMPERATURES ON BLOOD-PRESSURE AND PULSE-RATE IN A HEALTHY MAN OF TWENTY-SIX. (Riva-Rocci's sphygm., 15 cm.)

(From Müller, Deutsch. Arch. f. klin. Med., 1902, vol. lxxiv, Plate IV.)

The former studied the effect of cold hydrotherapeutic procedures on persons with mild neurasthenia, early phthisis, and other conditions in which a practically normal cardio-vascular state was present. All measurements were made with the Riva-Rocci apparatus (5 cm.), between 8 and 10 A. M. to avoid diurnal variations, and in the recumbent posture. He noted the pressure, pulse-rate, and temperature five minutes before the bath or douche, and five, fifteen, thirty, and sixty minutes after. He also used great care in observing, not only the temperature, but also the duration of the bath. In this way he found a definite relation between the duration of the cold and the change in pressure, and considers the divergent results of previous investigators as due to varying time or temperature employed. He found that full baths at 80° C. gave a rise in pressure in one-half minute; at 13° to 16° C. in one minute; at 20° to 22° C. only after two minutes or longer, a fall occurring when the bath was discontinued sooner. He could ascertain no constant relation between pressure and pulse-rate, or body temperature. The relation between duration and effect was always striking.

Müller, using a Riva-Rocci instrument with 15 cm. armlet, and apparently every precaution, came to the following rather striking conclusions: 1. Water baths of a temperature below 33° to 35° C. produce a rise in blood-pressure, with slowing of the pulse, throughout the whole duration of the bath. The colder the temperature the greater the rise, in the same individual. 2. Water baths from 33° to 35°, to about 40° C. (the so-called indifferent zone) produce, after a short initial rise, a fall in pressure to or below the normal, followed by another rise. The behavior of the pulse differs in this group; below 37° C. it diminishes, above it increases. 3. Water baths from 40° C. up produce a rise in pressure, lasting during the entire bath, with increase in pulse-rate. These effects are very readily seen in the accompanying charts (Fig. 41). 4. With hot-air and steam baths at 50° C., the same rise in pressure and pulse-rate occurred in normal persons. With certain sick patients, however, in whom heart weakness probably existed, the rise in pressure soon gave way to a fall, or the pressure fell from the start. The individual reaction to the varying temperatures employed was quite different in amount, but the same in kind, for all the cases studied. The more complicated hydrotherapeutic questions I will not enter upon, since we, in this country, unfortunately lack the requisite technical knowledge of the subject. Those interested may find a full literature in Müller's paper.

We may therefore conclude that, so far as our present knowledge goes, baths at about body-temperature are without marked effect on the blood-pressure; that cold baths produce a rise in systolic pressure, the extent of which, in any given individual, is greater the lower the temperature and the longer the period of exposure to it, within limits which undoubtedly exist, but have not yet been determined; that hot baths follow the same rule, differing only in that the rise in systolic pressure is accompanied by a more rapid pulse. Since the same phenomena have been observed with steam and hot-air baths, there seems no reason to doubt that the efficient cause is the change in external temperature. Of the actual mechanism by which the pressure changes are brought about, whether increase of peripheral resistance, or of the heart's energy, or both, I do not think we may form any valid opinion at present. Determinations of systolic and diastolic pressures are much to be desired in this connection.

K. Influence of Atmospheric Pressure.—There is little to be found on the effects of atmospheric pressure on arterial pressure in the normal man, the bulk of the observations being in conditions of shock. Lazarus and Schirmunski, however, amplified their animal experiments (see page 38), by subjecting men to a gradual reduction in atmospheric pressure from 760 to 380 mm. In all cases a fall of 25 to 35 mm. (v. B.) was obtained, and headache, dizziness, palpitation, or dyspnœa appeared in varying combination.

L. Influence of Menstruation.—I have found but three references in the literature to the behavior of arterial pressure during the menstrual period, in normal women. These do not allow of definite conclusions, because they fail to agree completely. We may only say that a diminution of tension may be normal.

Wiessner studied the question especially and claimed to find a regular fall of 20 mm. (R. R. 5 cm.) during menstruation, the normal level being restored three or four days after cessation of the flow. Hensen could not satisfy himself of any particular change. Rosse, who made measurements with the tonometer, in women who showed no particular disturbance during the period, as a check to his results in hysterical girls, noted either a slight diminution of tension or no variation. Federn noted a fall either shortly before, at, or after the beginning of the flow, the pressure rising gradually through the intermenstrual period.

M. Summary.—This array of facts regarding the variations of blood-pressure in the healthy man and their numerous causes might, at the first blush, lead one to conclude that, where such instability is normal, there can be no firm ground for the construction of a standard by which to gauge the abnormal. This is certainly untrue. Because the pulse-rate may reach 120 during exertion or excitement, the rapid pulse of fever is no less significant; nor is the existence of a digestion leucocytosis a valid reason for considering inflammatory leucocytosis a normal phenomenon. We must know the possibility of digestion leucocytosis that its occurrence may not lead us into an erroneous diagnosis. Similarly we must know thoroughly the causes for changes in blood-pressure during health; then we shall be able, by excluding or taking account of them, to recognize and value the pathological indications.

We have seen that mental activity, and especially excitement, produce the greatest rises in pressure. Exertion also causes a rise, the amount depending on the severity of the effort required. Mental and physical rest must therefore be secured in all clinical observations. Excessive changes in external temperature should be guarded against. Since posture may have some influence, it is wise to make all tests in the same position, either sitting or reclining; preferably the latter, because it conduces most to the desired rest. The diurnal variations, including the effects of eating and of sleep, must be in mind. In following cases from day to day, especially with reference to the effects of treatment on the blood-pressure, determinations should be made at the same hours. Everyone admits the importance of this in studying the course of the temperature in disease. With the arterial pressure it is not a whit less important. Where experiments are made which occupy only minutes or hours, instead of days, it is essential that a record of the pressure be made at frequent intervals for some time previous, to establish the normal level and the extent of the periodic variations. When this is done, it may be possible to demonstrate changes of small extent, which, lacking this standard for comparison, would be considered within the limits of normal variation.

Studied under such conditions, and they are scarcely more rigorous than the simple taking of temperature requires, it is surprising how constant the blood-pressure may be. Day after day it may not vary 10 mm. at a given hour. Hensen was struck with this, and I have found it conspicuous in measurements made on successive days in office practice, where the conditions do not vary. In hospital charts, where observations are made hurriedly, and so many causes for psychical irritation exist, I see more fluctuation.

From individual to individual the differences are more pronounced, but not more so than the differences in pulse-rate. The vast majority of healthy adults will show systolic pressures of 100 to 130 mm. (12 cm. armlet), 110 to 150 mm. (5 cm.). The diastolic pressure will be 25 to 40 mm. lower. After middle life 130 to 145 mm. (12 cm.) is not uncommon, 140 to 160 mm. (5 cm.). In childhood 90 to 110 mm. after the second year, 75 to 90 mm. before it, make the lower end of the scale, with probably a slighter difference between systolic and diastolic pressures. With women the pressure tends to be slightly lower, more definitely so when the 5 cm. armlet is used.

I regard with suspicion any pressure above 135 mm. (12 cm.), 150 mm. (5 cm.), in a young person, 145 mm. (12 cm.), or 170 mm. (5 cm.), in an older one, when found on several examinations and with due precautions. Above 145 mm. (12 cm.), 160 mm. (5 cm.), before middle age, or 160 mm. (12 cm.), 180 mm. (5 cm.), after it, I believe is definitely pathological, if constantly present as the average reading. For the lower limit of systolic pressure in health I would take 90 mm. (12 cm. and G.), 100 mm. (5 cm.), in adults, about 80 mm. in children.

I should like to lay stress on the importance of the simultaneous measurement of systolic and diastolic pressure in determining normal conditions. The difference between the two is ordinarily 25 to 40 mm. in my experience, the lower figures being more common in women. In old men 50 mm. may be found, an expression, in terms of pressure variation, of the well-known large pulse of the aged. A pulse which shows less than 20 mm. difference between systole and diastole I think is abnormally small, one over 50 mm. similarly large. When it is only possible to estimate one of the pressures, the systolic is the surer guide, because our data for it are more complete, and pathological changes affect it more than the diastolic.

LITERATURE

v. Basch, S. Die Herzkrankheiten bei Arteriosclerose, Berlin, 1901, p. 3.

Bruce, L. Some Observations upon the General Blood-pressure in Sleeplessness and Sleep. Scottish Med. and Surg. Jour., 1900, vol. vii, p. 109.

Bruck, Carl. Ueber den Einfluss kalter hydriatischer Prozeduren auf den Blutdruck. Inaug. Dissert., München, 1902, April.

Brush, C. E., and Fayerweather, R. Observations on the Changes in Blood-pressure during Normal Sleep. Am. Jour. of Physiol., 1901, vol. v, p. 199.

Carter, H. S. Clinical Observations on Blood-pressure. Am. Jour. of the Med. Sci., 1901, vol. cxxii, p. 854.

Colombo, C. Recherches sur la pression du sang chez l'homme. Arch. Ital. de Biol., 1899, vol. xxxi, p. 355.

Cook, H. W. The Clinical Value of Blood-pressure Determinations as a Guide to Stimulation in Sick Children. Amer. Jour. of the Med. Sci., 1903, vol. cxxv, p. 433.

Cook, H. W., and Briggs, J. B. Clinical Observations on Blood-pressure. Johns Hopkins Hosp. Rep., 1903, vol. xi, p. 451.

Cushney, A. R. Pharmacology and Therapeutics. Phila., 1903, pp. 139 and 271.

Doleschal. Vergleichende Untersuchungen des Gärtner'schen Tonometers mit dem v. Basch'schen Sphygmomanometer. Inaug. Dissert., 1900.

Edgecomb, W., and Bain, W. The Effect of Baths, Massage and Exercise on the Blood-pressure. Lancet, 1899, vol. i, p. 1552.

Ekgren, E. Ein eigenartiger Blutdruckbefund bei einem Falle von Mediastinaltumor. Fortschr. d. Med., 1902, vol. xx, p. 105.

Erlanger, J., and Hooker, D. R. The Relation between Blood-pressure, Pulse-pressure and the Velocity of Blood-flow in Man. Am. Jour. of Physiol., 1904, vol. x; Proceed. of Am. Physiol. Soc, p. xv.

Federn, S. Ueber Blutdruckmessung am Menschen. Wien. klin. Wochenschr., 1902, vol. xv, p. 835.

Gärtner, G. Ueber das Tonometer. Münch. med. Wochenschr., 1900, vol. xlvii,[2] p. 1195.

Goldwater, S. S. Notes on Blood-pressure in Man. Med. News, 1903, vol. lxxxii, p. 926.

Grebner, F., u., Grünbaum, R. Ueber die Beziehungen der Muskelarbeit zum Blutdruck. Wien. med. Presse, 1899, vol. xl, p. 2033.

Gumprecht. Experimentelle und klinische Prüfung des Riva-Rocci'schen Sphygmomanometers. Zeitschr. f. klin. Med., 1900, vol. xxxix, p. 377.

Hayaski, T. Vergleichende Blutdruckmessungen an Gesunden und Kranken mit den Apparaten von Gärtner, Riva-Rocci und Frey. Inaug. Dissert., Erlangen, 1901, Dec.

Hensen, H. Beiträge zur Physiologie und Pathologie des Blutdrucks. Deut. Arch. f. klin. Med., 1900, vol. lxvii, p. 438.

Hill, Leonard. On Rest, Sleep and Work and the Concomitant Changes in the Circulation of the Blood. Lancet, 1898, vol. i, p. 282.

Huber, A. Ueber Blutdruckbestimmungen. Cor. Bl. f. Schweiz. Aerzte, 1902, vol. xxxii, p. 425.

Jackson, J. M. A Few Remarks on Blood-pressure. Boston Med. and Surg. Jour., 1903, vol. cxlviii, p. 223.

Jarotzny, A. Zur Methodik der klinischen Blutdruckmessung. Centralbl. f. in. Med., 1901, vol. xxii, p. 599.

Jellinek, S. Ueber den Blutdruck des gesunden Menschen. Zeitschr. f. klin. Med., 1900, vol. xxxix, p. 447.

Kapsammer, Geo. Blutdruckmessungen mit Gärtner'schen Tonometer. Wien. klin. Wochenschr., 1899, vol. xii, p. 1279.

Karrenstein. Blutdruck und Körperarbeit. Zeitschr. f. klin. Med., 1903, vol. l, p. 322.

Kiesow, F. Expériences avec la Sphygmomanomètre de Mosso sur les changements de la pression du sang, chez l'homme, produits par les excitations psychiques. Arch. Ital. de Biol., 1895, vol. xxiii, p. 198.

Kornfeld, S. Erfahrungen über Trional als Schlafmittel mit besonderer Rucksicht auf die Beeinflussung des Blutdruckes. Wien. med. Blätter, 1898, vol. xxi, pp. 3, 25 and 40.

— Ueber den Einfluss physischer und geistiger Arbeit auf den Blutdruck. Wien. med. Blät., 1899, vol. xxii, pp. 635 and 668.

Larrabee, R. C. The Effects of Exercise on the Heart and Circulation. Boston Med. and Surg. Jour., 1902, vol. cxlvii, p. 318.

Lazarus und Schirmunski. Ueber die Wirkung des Aufenthalt in verdünnter Luft auf den Blutdruck. Zeitschr. f. klin. Med., 1884, vol. vii, p. 299.

Marey, E. J. La circulation du sang à l'état physiologique et dans les maladies, Paris, 1881, p. 334.

Masing, Ernst. Ueber das Verhalten des Blutdrucks des jungen und des bejahrten Menschen bei Muskelarbeit. Deut. Arch. f. klin. Med., 1902, vol. lxxiv, p. 253.

Moritz, O. Der Blutdruck bei Körperarbeit gesunder und Herzkranker Individuen. Deut. Arch. f. klin. Med., 1903, vol. lxxvii, p. 339.

Mosso, A. Sphygmomanomètre pour mesurer la pression du sang chez l'homme. Arch. Ital. de Biol., 1895, vol. xxiii, p. 177.

Müller, Otfried. Ueber den Einfluss von Bädern und Douchen auf den Blutdruck beim Menschen. Deut. Arch. f. klin. Med., 1902, vol. lxxiv, p. 316.

Potain, C. La pression artérielle de l'homme à l'état normal et pathologique, Paris, 1902.

v. Recklinghausen. Ueber Blutdruckmessung beim Menschen. Arch. f. exp. Pathol. u. Pharmakol., 1901, vol. xlvi, p. 78.

Rosse. Beitrag zur Blutdruckmessung bei Geisteskranken. Allgem. Arch. f. Psychiatrie, 1902, vol. lix, p. 737.

Schott, Theodore. On Blood-pressure under the Influence of Acute Overstraining of the Heart. N. Y. Med. Jour., 1902, vol. lxxv, p. 705.

Schüle. Ueber Blutdruckmessungen mit dem Tonometer von Gärtner. Berl. klin. Wochenschr., 1900, vol. xxxvii, p. 726.

Shaw, H. L. K. Arterial Tension in Childhood. Albany Med. Jour., 1900, vol. xxi, p. 88.

Sommerfeld, L. Blutdruckmessungen mit dem Gärtner'schen Tonometer. Therap. Monatschr., 1901, vol. xv, p. 72.

Swientochowski, J. Ueber den Einfluss des Alkohols auf die Blutcirculation. Zeitschr. f. klin. Med., 1902, vol. xlvi, p. 284.

Thayer, W. S. On the Late Effects of Typhoid Fever on the Heart and Vessels. Am. Jour. of the Med. Sci., 1904, vol. cxxvii, p. 391.

Tschlenoff. Ueber die Beeinflussung des Blutdruckes durch hydriatischer Proceduren und Körperbewegungen. Zeitschr. f. diät. u. physikal. Therap., 1898, vol. i, p. 328.

Weiss, Hugo. Blutdruckmessungen mit Gärtner's Tonometer. Münch. med. Wochenschr., 1900, vol. xlvii,' pp. 69 and 118.

Wiessner. Ueber Blutdruckuntersuchungen während der Menstruation und Schwangerschaft. Centralbl. f. Gynäkol., 1899, vol. xxiii, p. 1335.

Wren, A. A., and Oertel, H. A Note on the Physiological Action of Alcoholic Stimuli. N. Y. Univ. Bull. of the Med. Sci., 1901, vol. i, p. 184.

Zadek, I. Die Messung des Blutdrucks am Menschen. Zeitschr. f. klin. Med., 1881, vol. ii, p. 509.

CHAPTER VI

THE BLOOD-PRESSURE IN DISEASE

1. **Pathological limits of blood-pressure.**
    - A. Functional and essential elements of blood-pressure.
2. **High blood-pressure** (Hypertension).
    - A. Functional hypertension and its causes.
        - a. Physiological; pharmacological; toxic.
        - b. Acute cerebral compression and anæmia.
    - B. Essential or permanent hypertension.
    - C. Causes of essential hypertension.
        - a. In arterio-sclerosis.
        - b. In renal disease. Theories of cardiac hypertrophy.
        - c. In angio-sclerosis.
    - D. Effects and dangers of hypertension.
        - a. For the heart.
        - b. For the arteries.
3. **Low blood-pressure** (Hypotension).
    - A. Causes of hypotension.
        - a. Wasting diseases.
        - b. Drugs; infections; hæmorrhage.
        - c. Terminal hypotension.
    - B. Collapse and shock.
        - a. General features.
        - b. Experimental evidence.
        - c. Practical results.
    - C. Effects and dangers of hypotension.
4. **The relation of blood-pressure to pulse-rate and temperature.**
5. **Blood-pressure as a guide to the functional ability of the heart and the adequacy of the circulation.**

    Literature.

CHAPTER VI

THE BLOOD-PRESSURE IN DISEASE

1. PATHOLOGICAL LIMITS OF BLOOD-PRESSURE

When we turn to the enormous variations in blood-pressure seen under pathological conditions, the smaller physiological fluctuations become far less conspicuous. The agonal pressures of 40 mm. (R. R. 5 cm.) reported by Hensen, and Cook and Briggs's case of cerebral hæmorrhage with about 400 mm. (R. R. 5 cm.), represent the extremes of tension recorded by reliable observers in the literature. The latter figure must contain a very large elasticity error, due to the narrow armpiece, but the absolute value could scarcely have been below 300 mm. The more common readings in high tension cases lie in the neighborhood of 200 to 220 mm. (12 cm.), 220 to 260 mm. (5 cm.).

The upper limit to blood-pressure is set by the reserve force of the heart (see page 15) and, under ordinary circumstances, by the depressor mechanism. Such enormous pressures as 350 to 400 mm. have never been reported except as very temporary manifestations in cerebral compression. I have seen a number of cases, however, in which a pressure between 250 and 300 mm. (R. R. 5 cm.) was present at every examination during more than a year. Individual differences in the anatomical and functional state of the cardiac muscle will determine the extent to which, in any given case, increase of peripheral resistance will raise the arterial pressure.

Exact data concerning the minimum arterial pressure at which the capillary circulation can be maintained sufficiently to support life are scarce. Experimentally, the complete loss of vaso-motor tone which follows destruction of the spinal cord is followed by death (see page 18). In man such total vasomotor paralysis is probable only in fatal surgical shock and in the death agony after acute infectious disease. Hensen places the lowest systolic blood-pressure at which a continuance of the circulation is possible at 75 mm. (R. R. 5 cm.), about 55 mm. absolute value. All pressures under 100 mm. (R. R. 5 cm.) he considers subnormal. Cook and Briggs give one chart, from a case of traumatic shock which recovered, with pressure below 50 mm. for a brief period; 60 mm. they find usually of serious import. Kapsammer gives 60 mm. (G) as the lowest pressure compatible with life, but cites one case in which a woman lived thirteen hours after a severe fall with a tonometer reading of 40 mm. John found 35 to 40 mm. (G) in advanced phthisis a few days before death. These figures, in the light of Gärtner's own refusal (see page 68) to measure below 70 mm. with the tonometer, seem of doubtful worth. In my personal experience, pressures below 75 mm. (12 cm.) have been very rare, except momentarily during surgical operations, where I have seen below 40 mm. (J. 12 cm.).

For diastolic pressure it is difficult to give any figures. I have never found it above 200 mm. (Erl. 5 cm.), nor, since the use of the wide armlet, above 180 mm.; but I have not examined cases of cerebral compression. As the elasticity of the arterial wall diminishes rapidly with increasing pressure after the higher tensions have been reached (see page 25), systolic pressure tends to increase much more than diastolic under such conditions. For instance, a systolic pressure of over 220 mm. will commonly be associated with a diastolic some 60 to 80 mm. lower, instead of the 25 to 40 mm. usual at normal tensions. This large amplitude of the pulse-wave is readily seen in sphygmographic tracings, and, when one's attention has been drawn to it, becomes equally evident to the finger, if sufficient force is exerted upon the artery. The latter precaution is necessary, because excursion of the already tense arterial wall is slight, unless the pressure upon its two sides be nearly equalized.

For the lower limit of diastolic pressure I can give no figures. My own observations have been along different lines and I know of no accurate measurements in the literature. The Hill and Barnard instrument, as heretofore used, has given an indeterminate and intermediate pressure (see page 72).

A. Functional and Essential Elements of Blood-pressure.—We have seen (see page 18) that the vaso-motor tone compensates for the disproportion between the total blood-volume and the size of the vessels it is called on to fill. The lower limit of blood-pressure represents the pressure required to overcome the resistance of the arteries due to this necessary vaso-motor tone. We may speak of this as the essential portion of the arterial pressure, without which no circulation is possible. It must vary somewhat from person to person, but probably never amounts to much less than 50 mm. Hg. Such a minimum, however, is evidently much below the normal pressures of daily life. This difference is due to the changing tone of the small arteries throughout the body necessary to the functional activity of the various organs. We may speak of the excess of normal pressure over the essential as the functional element of blood-pressure. This varies from moment to moment and is especially dependent on the resistance in the splanchnic circulation. Changes in blood-distribution may occur without any variation in the total peripheral resistance, since dilatation of one set of arterioles usually causes reflex constriction of other remote ones; but such compensation is seldom perfect. In muscular exertion, for instance, we have seen that pressure usually rises. The functional element of blood-pressure, then, will vary with changing blood-distribution, especially with the demands for blood on the part of the brain, which is more dependent on general blood-pressure than the other organs.

In states of continued high blood-pressure due to a permanent increase in the resistance of the arteries, the essential element of pressure must rise also. This is an important practical consideration, for a fall to near the essential amount is dangerous to life per se. Where the average level of blood-pressure has been permanently above 200 mm., the lower limit must be above 75 mm. Hensen cites a case of chronic nephritis with an agonal pressure of 180 mm.

Undoubtedly a continued fall in pressure is far more significant of danger than any actual numerical value, as Cook and Briggs insist. Here the need of a graphic record is manifest.

2. HIGH BLOOD-PRESSURE (Hypertension).

By far the most valuable results of clinical sphygmomanometry have been in conditions of high arterial tension, and for a double reason. First, our previous methods for the discovery of increased blood-pressure (palpation of pulse, sphygmograph, detection of cardiac hypertrophy or accentuated aortic second sound) were frequently either inadequate or misleading; thus our diagnosis has gained accuracy. Second, the existence of continued high pressure has far greater absolute significance than the finding of normal or subnormal tension.

Objection may be made to the above statement, on the ground that the existence of a hypertrophied heart and ringing aortic second sound are our most valid evidences of the existence of hypertension. I will not dispute their validity. The difficulty is, that high blood-pressure is most common at the period of life in which obesity or emphysema frequently make the percussion and auscultation of the heart well-nigh impossible. It is in just such persons that the sphygmomanometer comes to our help.

As to the sphygmograph, few physicians will now claim for it much practical value, though its educational effect has been considerable. Apart from the fact that one may obtain almost any type of trace from any pulse, by varying the spring-pressure, the forms of pulse-curve generally pictured as indicative of high tension are not truly so. The trace with a gradual descending limb is usually given, as by Sansom.[1] This particular form of pulse, as my combined sphygmographic and sphygmomanometric work taught me, is regularly obtained when one narrows an artery almost to the point of obliteration. Thus it is evidence of a contracted artery, which may go with low tension, though frequently found with high.

The difficulty of estimating pulse tension by the finger has been alluded to (see page 44). Krehl[2] admits that the distinction between internal tension and arterial thickening may elude even the most experienced.

A. Functional Hypertension and its Causes.—a. Physiological; Pharmacological; Toxic.—Any condition in which increased blood-pressure is present, as a result of a temporary cause acting on the heart or vessels, we may speak of as evidencing functional hypertension. The rise in pressure due to excitement or exertion in the normal man falls under this heading. The marked augmentation of tension during true labor pains is due to these physiological causes, plus compression of the abdominal vessels, which drives a large volume of blood to the heart. In certain nervous affections hypertension occurs, apparently from reflex vaso-constriction. In melancholia it closely parallels the intensity of mental anguish, the stimulus which calls forth the reflex originating in the psychical sphere.

---

[1] Sansom, p. 418.

[2] Krehl, L. Die Erkrankungen des Herzmuskels, etc., p. 374.

In tabes, the painful visceral crises have a similar association with heightened vaso-motor tone, the stimulus coming from spinal or peripheral neurons. Acute pain of other kinds, biliary colic for instance, may cause considerable hypertension. The rise in blood-pressure at the onset of acute peritonitis must also be due to a reflex vaso-constriction. The various drugs which stimulate the circulation produce functional hypertension. The digitalis series act preeminently by increasing cardiac energy, secondarily through vaso-constriction; adrenal extract produces its remarkable effects in the opposite order; strychnin and ergot raise pressure only by arterial constriction. Camphor and caffein, as well as atropin, increase cardiac energy, and peripheral resistance also. This, of course, applies only to therapeutic doses. Nicotin, in animals, produces a great increase in blood-pressure, and hypertension in smokers has already been mentioned. High pressure is frequent in lead poisoning and is said to be associated with acute gout. The cause is probably a toxic one in both cases. In many cardiac patients one finds the pressure somewhat above the normal from time to time, quite independently of the manner in which the heart is acting, therefore undoubtedly due to complex peripheral influence. In none of these conditions does the systolic pressure often pass 200 mm. (R. R. 5 cm.), 175 mm. (12 cm. or G). A much greater hypertension, from increased activity of the vaso-constrictor centres, is the asphyxial rise already considered. This is met with clinically in obstructions of the upper air-passages, and, in a less degree, when more chronic respiratory or cardiac diseases lead to deficient oxygenation of the blood. Toxic hypertension of extreme grade is seen in uræmia and eclampsia, because the large functional rise is superimposed on an existing essential hypertension.

b. Acute Cerebral Compression and Anæmia.—The highest arterial pressures ever recorded in man have accompanied acute compression of the brain, in fracture of the base of the skull and apoplexy. An understanding of the real causes of this extreme functional hypertension is of the first importance, if one would intelligently utilize the indications of the blood-pressure for accurate diagnosis and treatment. The theoretical basis is now secure in its main features, and makes one of the most fascinating chapters in experimental pathology. Its fullest details may be found in the monograph of Kocher, and it is also most interestingly presented in English by Hill, and by Cushing, both of whom have made important contributions to its elucidation.

Acute cerebral anæmia, that is, the rapid and complete cessation of the cerebral circulation, as Hill has shown, produces symptoms identical with complete asphyxia. The usual sequence is: A. Loss of consciousness. B. Respiratory spasm. C. Slow heart and rise of blood-pressure with cessation of respiration. D. Fall of blood-pressure, acceleration of heart, and death. This indicates distinctly that the bulbar centres are first excited and then paralyzed. If the animal be in a state of shock, or the anæmia be slow in onset, the excitatory symptoms fail to appear. During the development of these events, while the anæmia is partial, marked Traube-Hering blood-pressure waves and Cheyne-Stokes respiration are common.

Cerebral compression produces the same bulbar symptoms as acute anæmia. This seems paradoxical until one remembers the mechanical limitations of the cerebral circulation. Since the cranio-vertebral cavity is closed and the brain substance incompressible, the total amount of blood in the brain is invariable, except for the slight expansion made possible by increased absorption of cerebro-spinal fluid at high pressures. If, then, a foreign body of any kind be introduced into the cranial cavity, room can only be made for it at the expense of the blood in neighboring veins and capillaries. Compression therefore produces local anæmia, and the symptoms are due, not to the pressure, but to the cessation of blood-flow. At first only the veins will be narrowed and capillary pressure raised. In Kocher's nomenclature, this is the stage of compensation. Few symptoms occur. Little increase of pressure is required to bring about venous stasis, with much diminished capillary flow, and brain tension equal to the arterial. In this second period, Kocher's initial stage of manifest cerebral compression, numerous subjective symptoms of general cortical or cerebellar anæmia appear; headache, vertigo, etc. The ophthalmoscope shows the picture of choked disc. If the local compression be over an extensive area, or situated in the posterior fossa; or, if general cerebro-spinal compression be produced, as in Cushing's experiments, by introducing fluid into the cranium, the circulation in the medulla will be affected. The bulbar symptoms at this period are, slowing of the pulse, from stimulation of the vagus centre, and some rise in pressure, from vaso-motor stimulation. The latter is not constant.

Any further increase in pressure will now empty the veins and capillaries, and, as soon as it passes the level of arterial pressure, will absolutely cut off the circulation. This third advanced stage of manifest cerebral compression is, in reality, an acute cerebral anæmia, and leads to absolute loss of function. Now it is that the medulla responds with an effort to preserve its life. The vaso-motor centre automatically raises general blood-pressure above the intracranial tension, and its blood-flow begins anew. If the compression goes higher, the vaso-motor centre follows with another rise, and so, step by step, the blood-pressure may keep just ahead of advancing brain-pressure until such enormous figures as 300 mm. and more are reached. Each rise is not steadily maintained, however, but in many cases seems to overcompensate and be succeeded by a fall, then a rebound, as anæmia of the bulb recurs, in pendulum-like oscillation. These are the pronounced Traube-Hering waves, which may be appreciated in making sphygmomanometric readings. The respiratory centre shows a similar rhythmical variation in its activity, deep breathing coinciding with the period of high blood-pressure and established circulation, apnœa with its interruption; the clinical Cheyne-Stokes respiration. The vagus centre is likewise stimulated, and the pulse-rate falls materially, as every one has noticed in apoplexy. This in part antagonizes the good effect of vaso-motor activity; at least the most complete parallelism between brain- and blood-pressures is found after section of both vagi. The complete chain of events is beautifully shown in Cushing's tracings.

If the intracranial tension be too long maintained, or go too high, the medullary centres become exhausted and the final paralytic stage sets in, an exact counterpart to the terminal period of cerebral anæmia or asphyxia. The blood-pressure falls, with rapid, running pulse, and death soon ensues. I have gone thus at length into the experimental findings, because the clinical picture is their perfect duplicate. This will receive consideration later, particularly with respect to surgical treatment. Too great emphasis cannot be laid on the fact that the rise in blood-pressure during acute cerebral compression is absolutely essential to the preservation of life. On the other hand, it cannot be denied that, where the cause of the increased intracranial tension is a hæmorrhage, the hypertension augments it, so that a vicious circle is established. The same may occur when the primary cause leads to œdema, with secondary increase of compression, the transudation being hastened by the greater arterial tension, so long as the venous pressure remains high.

B. Essential or Permanent Hypertension.—A permanently high level of blood-pressure is found in connection with a lasting increase in the peripheral resistance which the left ventricle must overcome, combined with hypertrophy of the latter, by which it is enabled to meet the demand. Such changes belong to the diseases of the arteries themselves, which we include under the general term arterio-sclerosis, and to the cardiovascular lesions of kidney disease. An apparent hypertension, that is, a high systolic, but not mean or diastolic pressure, is seen in aortic insufficiency.

C. Causes of Essential Hypertension.—In the normal animal or man the average level of blood-pressure is maintained with great constancy. The complex nervous mechanisms, which coördinate the activities of the cardiac and vascular muscles, seem to be directed toward securing the greatest local variations in blood-flow with the least alteration of general arterial pressure. We have seen that the largest part of this regulating function is lodged in the splanchnic circulation (see page 22). If the blood-pressure rises to the point where the heart is embarrassed, reflex dilatation of the countless arterioles of the abdominal viscera, evoked by the depressor nerve, promptly causes a fall. It seems evident, therefore, that an abnormally high pressure cannot exist permanently, unless there has been some damage to the regulating power of the visceral circulation.

But the increase in peripheral resistance of itself would be insufficient to account for a lasting increase in aortic pressure. The depressor mechanism is twofold, and the normal heart shields itself from overstrain by another reflex through this nerve, which automatically slows it by way of the vagus. This probably only comes into play in the more considerable and sudden variations of pressure. It evidences, however, a limit to the reserve force of the heart. With increase in peripheral resistance, increased cardiac energy is required to keep the same volume of blood flowing through the capillaries, and, if it is lasting, the working ability of the heart must be raised to a higher average level. This necessitates either more or larger muscle fibres. Hence, permanent high blood-pressure cannot be maintained without hypertrophy of the left ventricle.

a. In Arterio-sclerosis.—When we leave the domain of theory for the world of observed facts, we find the most complete confirmation of these conclusions in the case of arterio-sclerosis. In many cases presenting evident thickening of the superficial arteries, high blood-pressure and the signs of an enlarged left ventricle, with ringing aortic second sound, concur. Many cases in my experience, however, have failed to show increased tension with the sphygmomanometer, and the literature abounds with similar reports (see Arterio-sclerosis, Chapter VII, 1, C.), which have been a stumbling-block to many. Autopsy observations coincide with this, cardiac hypertrophy often failing, in spite of well-developed arterio-sclerosis. The explanation of this lies in the local distribution of the process. The arterial changes, as all pathologists know, affect different portions of the vascular system in a most irregular way. From the physiological considerations, we should not expect an effect on general blood-pressure, or on the heart, where the splanchnic arteries have not suffered. This has been beautifully substantiated from the anatomical findings by Hasenfeld, and by Hirsch. In careful investigations of the comparative weights of the separate portions of the heart and the body weight, by Müller's method, they proved definitely that: Arterio-sclerosis leads to a hypertrophy of the left ventricle only when the splanchnic arteries or the aorta above the diaphragm are highly diseased. The arterio-sclerosis of the remaining vascular districts does not appear to exert this influence. The correspondence between this and the results of ligature of the same vessels in the research laboratory fifty years before, is good proof of the fundamental importance of physiological knowledge in clinical medicine.

b. In Renal Disease. Theories of Cardiac Hypertrophy.—With the hypertension of renal disease the facts admit of less ready explanation. Bright, in 1836, first recognized the coincidence of a hypertrophied heart and alterations in the kidney, with the clinical picture which bears his name. He postulated an altered composition of the blood as the cause, which he said might either stimulate the heart abnormally, or increase the resistance in the small vessels and capillaries. Though many new facts have been adduced, no theories have yet been deduced which are much nearer the truth.

The older explanations, from Traube to Cohnheim, were purely mechanical, referring the hypertrophy, either directly or indirectly, to increase in total blood-volume, or to increase in resistance due to the narrowing of the vascular stream-bed in the kidneys. Neither of these are tenable in the light of physiological experiment, or of clinical fact. Johnson first introduced the chemical standpoint, supposing that the accumulation of waste products in the blood causes vaso-motor spasm, and secondary hypertrophy of the muscular coat of the arteries. O. Israel showed that the accumulation of nitrogenous waste (urea) in guinea pigs might produce hypertrophy of the heart, and looked on this as due to a direct stimulation. He considered the increased work of the heart a compensatory mechanism, counteracting the deficiency of renal parenchyma by supplying the remaining portions an increased amount of blood. Especially striking in his work was the absence of any significant change in blood-pressure, despite the cardiac hypertrophy. This seems to me evidence that in clinical, as opposed to experimental nephritis, damage to the splanchnic circulation must be an essential factor. Gull and Sutton went to the opposite extreme and looked on the whole development of nephritis as secondary to a general change in the arteries, which they euphoniously named "arterio-capillary fibrosis." Theories of nervous and other origins have also been brought forward, but not with the same influence on medical opinion as those mentioned.

Certain newer facts with relation to the cardiac hypertrophy of Bright's disease must be mentioned. The most important is, that post-mortem statistics have proven in most cases the hypertrophy of the whole heart, auricles and right heart, as well as left ventricle. Bamberger's figures showed this, and the careful tabulations of Hirsch, by Müller's method, confirm it. The latter, however, in the early stage of contracted kidney, saw only the left ventricle enlarged. Even Senator, who lays much stress on the hypertrophy of the portions of the heart which are not directly affected by increased aortic pressure, considers it proven that the hypertrophy of renal disease affects the left heart, either exclusively or to a disproportionate degree. He believes that, in contracted kidney, pure concentric hypertrophy is found; whereas excentric, or hypertrophy with dilatation, is the rule in chronic parenchymatous nephritis. Adami, on the other hand, disputes the existence of pure concentric hypertrophy under any circumstances, from observation and theoretical grounds alike. Other facts of importance are: the great frequency of splanchnic arterio-sclerosis in renal disease, a matter of common observation and especially noted both by Hasenfeld and Hirsch; also the appearance of high blood-pressure in acute nephritis at a very early period, cardiac hypertrophy not occurring until much later, and requiring at least one month for its development. Traube first called attention to this, Riegel later.

Senator's theory distinguishes between the origin of the hypertrophy in contracted kidney, and in other forms of nephritis. In the latter, acute and chronic parenchymatous nephritis especially, he considers there is a more active irritant, and more retention of waste products. The vessels outside the kidney, as well as the latter itself, are damaged by the irritant, and this causes the œdema, which for a time removes some of the irritant and excretives from the blood. If the renal lesion heals, the irritant has disappeared, œdema is absorbed, and heart and vessels again become normal. If the process becomes chronic, the continued slighter irritation and accumulation of waste act as constant stimulants to heart and vessels, causing hypertrophy of the former and thickening of the latter. Since the systemic circulation is under a much higher relative pressure, the left ventricle must be especially affected. In chronic interstitial nephritis he presupposes a much weaker irritant, affecting kidneys, heart, and vessels simultaneously. Nitrogenous excretion is not diminished until late, when much renal epithelium has been lost; then a further increase in pressure occurs. In consequence of the heightened pressure, more blood will pass through the kidney, and the urinary water and salts will be increased. The supposed irritant probably arises outside the kidney.

Within the last few months Erben has proposed that we should regard the albumin hunger of the cells, called forth by the albumin deficiency in the blood, as the primary cause of the rise in blood-pressure and cardiac hypertrophy; the mechanism of its action being partly through the vaso-motor system, partly by increased work of the heart to supply the demand for more nutritive material. Since all the previous researches collected by Askanazy, as well as his own studies on a large material, show a normal blood composition and entire absence of hydræmia in the types of nephritis without œdema—the cases in which the highest blood-pressure is seen—this theory does not appear to hold water.

From the number of these theories it is obvious that no single explanation can harmonize all the observed facts. Either new light must come which will unify our present evidence more completely, or our generalization must be less extended and recognize the coincident action of several causes, now one and now another preponderating. From the standpoint of the blood-pressure, not of the cardiac changes, it seems to me that an increase in peripheral resistance, together with some loss of the regulating function of the visceral circulation, must be assumed. O. Israel's experiments, for instance, in which artificially produced nephritis led to cardiac hypertrophy, did not show an appreciable rise in pressure. Here the vessels were evidently unaffected. The hypertrophy, but not the blood-pressure, went hand in hand with the loss of secreting renal substance. In human nephritis such a definite relation does not exist. Besides, with all due allowance for the undoubted hypertrophy of the whole heart in contracted kidney, it seems to me that overmuch stress is laid on it, from the small number of weighings by Müller's method, compared with the impression of pathologists from many thousand autopsies. Even Hirsch admits that the early cases do not show it, and that left ventricular hypertrophy predominates in all cases. Observations for a number of years will be necessary to clear up this point, and systematic blood-pressure records on all nephritic patients will add greatly to the value of the post-mortem findings, which allow us to judge of physiological alterations only by inference.

Whether the increased resistance and the loss of splanchnic regulation are one and the same, that is due to narrowing of the visceral circulatory area, either through vaso-motor spasm or anatomical change; or whether some of the increased resistance lies outside the abdominal vessels, is another problem. Again careful pressure observations during life will help us. Krehl[1] thinks continued spasm a priori unlikely. Hirsch and Beck were unable to prove increased viscosity of the blood in the majority of cases of nephritis they examined, but it was markedly greater than normal in three cases of genuine contracted kidney. More studies of this, and of the osmotic pressure of the blood, are to be desired, since general peripheral resistance might thus be raised. It is also conceivable that increased blood-volume, which in the presence of a normal vaso-motor mechanism is powerless to raise pressure, may contribute to this end when the regulation is disturbed. As a factor in chronic interstitial nephritis it cannot be considered, but in the acute forms, where actual retention occurs, it may be significant, like Erben's albumin hunger of the cells. Certainly contemporary opinion inclines toward the primary stimulation of the heart as the cause of its hypertrophy. I am quite willing to admit this as one factor, especially in the various forms of parenchymatous nephritis. I also think that the hypertrophy of the whole heart in chronic Bright's, which appears late, probably owes its origin to this cause; the retention of waste products, which occurs during the latter part of chronic cases being the possible stimulant. Nevertheless, for the reasons already given, I cannot abandon the opinion that increased resistance and diminished splanchnic compensation are essential hypotheses for the comprehension of the arterial hypertension, in the light of our present knowledge, whatever be the source of the irritant which evokes them.

---

[1] Krehl. Pathol. Phys., p. 37.

c. In Angio-sclerosis.—One other class of cases with essential hypertension remains to be considered. It is entirely a clinical group, and one which depends for its differentiation largely on the sphygmomanometer. In it are comprised all those persons who show permanently high blood-pressure, but neither discoverable arterial thickening, nor any clinical evidence of nephritis after repeated examination, not only as to the presence of albumin and casts, but with reference to the quantity of water and solids excreted as well. v. Basch has done the greatest service through insisting on the recognition of this condition, which he calls angio-sclerosis. While I consider many of his cases of so-called "manifest angio-sclerosis" as undoubted chronic nephritis, and am not convinced as to many of his pseudo-type, there remains the large group, which he names "latent angio-sclerosis." Huchard has a similar picture in mind in his "presclerosis." This condition, high blood-pressure, some cardiac hypertrophy, and symptoms referable as a rule to relative insufficiency of the heart, in middle-aged or elderly persons, who give no evidence of peripheral arterio-sclerosis or chronic Bright's, is comparatively frequent in my experience. I am convinced that its recognition and treatment are important. The special consideration of it will follow later. As to its cause, early sclerotic changes in the splanchnic vessels, as maintained by v. Basch, seems the most probable.

D. Effects and Dangers of Hypertension.—Hypertension is not merely a symptom of diagnostic and prognostic value, nor is it to be considered only as an effect of causes acting on the heart and vessels. It is of itself a source of altered function throughout the circulatory system, which leads to further secondary changes. These cannot in all cases be clearly separated from the primary changes producing the high pressure, but they may frequently be distinguished anatomically as well as theoretically.

a. For the Heart.—For the heart, increased pressure necessitates increased work to maintain the same intensity of capillary circulation. Of greater importance than the need for absolute increase of cardiac energy, is the relatively greater increase in the demands during muscular exertion. The mechanical disadvantage of increased peripheral resistance is the same in kind as that of narrowing the aortic orifice. Benno Lewy [1] has demonstrated for this, as for the other obstructive lesions, the extreme grade to which the necessity for a larger blood-flow during exertion augments the work of the heart. Since this additional output may raise the heart's work as much as thirteen-fold, it is evident that hypertension, like valvular lesions, will sooner lead to failure of compensation in those whose occupations are laborious.

The influence of nutrition is equally potent. It is well known that hypertrophy cannot occur unless the heart-muscle is well-nourished (Krehl, Benno Lewy, Rosenbach, etc.). Therefore accommodation to hypertension is apt to be more complete in the young and vigorous and fail most conspicuously when wasting disease is present (see Sibson's cases). Most important, however, as a source of danger, is the occurrence of structural alterations in the wall of the heart. Hasenfeld and Romberg showed in healthy dogs, that hypertrophied heart-muscle may possess the same reserve force as the normal. Nevertheless, as Krehl [2] says: "It can, but unfortunately it usually does not. In reality the old dictum of medicine remains justified: A hypertrophic heart is not so good as a normal one." Whether we consider the damage to the hypertrophic muscle as due to degenerative changes, as did the older pathologists; as secondary to sclerosis of the coronary arteries (Krehl,[3] v. Basch, Huchard's cardio-sclerosis); or adopt the most recent view of Albrecht, that chronic interstitial and parenchymatous inflammatory processes are an integral part of the hypertrophic process; at least we may feel sure that, sooner or later, the accommodative power of such a heart will become insufficient for the demands upon it.

The degree of hypertension and the time of development of the cardiac insufficiency have no relation to one another. v. Basch, in his book, has brought this out admirably, and his findings coincide with my experience. An increasingly high blood-pressure is a much more serious sign than any absolute value, especially when treatment fails to influence it. With the development of symptoms from the side of the heart the pressure may fall, or it may even rise; in the latter case indicating a still increasing peripheral resistance; in the former, of course, a diminution in cardiac energy.

---

[1] Lewy, Benno. Zeitschr. f. klin. Med., 1897, vol. xxxi, p. 532.

[2] Krehl, L. Pathol. Physiol., p. 51.

[3] Krehl, L. Erkrank. des Herzmuskels, p. 350.

The most common complaints one meets with in this condition are: dyspnœa on exertion, or the more severe spontaneous attacks, which we call cardiac asthma; precordial distress, varying in degree from slight substernal oppression or a feeling of tightness, to the most intense angina pectoris; and subjective or objective disturbances of the heart's rate or rhythm. Attacks of œdema of the lungs are rarer and depend on the sudden development of weakness of the left ventricle, throwing more work on the right than it can perform.[1] If the right ventricle gradually becomes secondarily insufficient, then general venous congestion and œdema set in. Death may come suddenly in an anginal seizure, or an attack of cardiac asthma or pulmonary œdema; or it may be the result of gradual asystole.

b. For the Arteries.—Not the heart alone suffers from the effect of continued high blood-pressure, but the arteries as well. The elastic distensibility of the arterial wall decreases with increasing tension. The proofs of this have been cited (see page 25). The immediate effect on the character of the pulse, a greater systolic elevation of pressure, has been explained. Thus, in all high tension pulses, where the circulation is at all adequate, one finds a difference of 50 to 80 mm. between systolic and diastolic pressures. The continued overstretching of the walls of the arteries leads in many cases to a general dilatation of the larger vessels, which are subject to the greatest systolic strain. The most straightforward examples of this are seen in aortic insufficiency, where the systolic elevation of pressure is sudden and extreme, but the general mean is not markedly altered. Every clinician must have seen examples of such dilatation of the abdominal aorta, carotid, subclavian, brachial, radial, and femoral arteries, as a result of aortic regurgitation consecutive to rheumatic endocarditis in young people, where primary arterial disease is practically excluded. Hasenfeld and Romberg noted a similar dilatation in animals with experimental aortic leakage. I have had evidence, both by physical signs and fluoroscope, of a dilated ascending aorta, which simulated aneurism, in two such cases. In chronic nephritis, a dilated aorta is not infrequent at autopsy, but here arterial disease as a primary factor is harder to eliminate. Certainly the mechanical moment is an efficient aid in its production, as of the more general dilatations, which are also found in patients with Bright's or some forms of arterio-sclerosis.

---

[1] See Cohnheim and Welch. Ueber acutes Lungenœdem. Cohnheim's Gesam. Abhandlungen. Berlin, 1885, p. 593.

The smaller arteries may undergo dilatation as a result of the increased internal tension to which they are subjected, a late event of degenerative changes in their coats, due to general arterial disease.[1] Rosenbach believes that with increased blood-pressure the arterial walls hypertrophy, because of the extra work they are called on to perform and the greater blood-supply which he considers they receive; the later degenerative changes he calls decompensative, thus classifying practically all the arterio-sclerotic changes in the arteries, as opposed to the arterioles, as secondary to hypertension. The hypertrophy of the muscularis, described by Johnson, lends itself to a similar explanation; but so good an observer as Senator[2] thinks it a chance find. He insists that intima and adventitia alone show constant increase in thickness in chronic nephritis. Recent experiments by Sumikawa are of special interest. He tied off a considerable portion of artery and subjected it to high internal pressure, by the injection of salt solution. The rupture of some of the elastic fibres, thus produced, was followed by proliferation of the media, and of the intima to an even greater degree. We may therefore say that the differentiation of the complex arterial lesions, and of the order of their development is impossible to-day; but that many of them are of a secondary nature and, in certain of their features, due to increased internal tension, seems assured.

The diminished distensibility of the arteries, and the sharp rise in pressure at every systole of the ventricles, introduces another danger, that of rupture. We know that the healthy artery can stand a strain which the most extreme hypertension never approximates. Given the degenerative changes, however, which are common in diseases associated with high blood-pressure, plus the sudden augmentation of tension which excitement and exertion cause, and rupture becomes probable. Experience bears this out. Of Bright's original cases a large proportion died of apoplexy, and the occurrence of cerebral hæmorrhage as a common termination of Bright's disease has been well established. All the recent literature of clinical sphygmomanometry confirms it, from the standpoint of high arterial pressure. Of course the real cause lies in the local weakening of a cerebral vessel. One must not consider that height of blood-pressure is any index of the probability of an apoplectic seizure, save in a general way.

---

[1] See Krehl. Pathol. Physiol., p. 108, and Erkrank. des Herzmuskels, p. 339.

[2] Senator. Erkrankungen der Nieren, p. 118.

The other dangerous condition due to giving way of the arterial wall, namely, aneurism, also depends primarily on the local lesion, secondarily on the general pressure. One sees a number of cases where high tension is not in evidence. When present, however, it is one more element in the already unfavorable prognosis. Nose-bleed is a minor manifestation of the same tendency.

3. LOW BLOOD-PRESSURE (Hypotension)

For practical purposes we may draw the line between low normal and subnormal systolic pressure at 100 mm. (R. R. 5 cm.), 90 mm. (12 cm. and G). Of course the real dividing line is an individual one, to determine which we should have previous records of the same patient in health. This is seldom possible, and we must take the lowest normal as our standard, just as we do for the leucocyte count. Sex, age, occupation, and muscular development may also be taken into account in our judgment, if not put into definite figures. One may at any rate be sure that 70 mm. is very marked hypotension.

A. Causes of Hypotension.—a. Wasting Diseases.—In medical practice, hypotension is commonly seen in wasting diseases and cachectic states, such as advanced phthisis, carcinoma of the stomach, and general paralysis of the insane. If anything could be called essential hypotension, this form would most justly deserve the name, for it seems to me unquestionably associated with a reduction in all three factors concerned in maintaining the blood-pressure. All the life processes are carried on at the lowest possible level, that is, the functional element of peripheral resistance is reduced to its lowest terms. The brown atrophy of the heart, found post mortem in these very conditions, bespeaks a diminished output of cardiac energy; and the reduction of total blood-volume is equally shown by the small and empty pulse during life, and the bloodlessness of the tissues after death.

b. Drugs; Infections; Hæmorrhage.—Temporary falls in pressure result from certain drugs which cause vaso-dilatation, the nitrites especially. Poisonous doses of drugs, which paralyze the vaso-motor centre or heart, naturally result in a fatal fall. Chloroform is the most important of these, and low arterial pressure occurs early in chloroform anæsthesia and needs careful watching. Various infections and toxæmias may be accompanied by hypotension in some persons, and are most apt to be when severe. Typhoid fever is more frequently hypotensive in the average case than the other acute diseases, pneumonia least. Hæmorrhage, when profuse, or occurring in persons whose vaso-motor centres have lost their normal function, as in shock or severe infections, leads to a rapid reduction of pressure.

c. Terminal Hypotension.—It goes without saying that the process of dissolution is attended by a fall in blood-pressure to the zero mark, irrespective of which particular leg of the tripod of life gives out first. The rapidity with which this terminal or agonal hypotension develops, differs much. In sudden cardiac death, as seen in aortic disease or angina pectoris, or in enormous hæmorrhage, it comes almost instantaneously. After lingering illness, pressures below 60 mm. (R. R. 5 cm.) may persist for several days before the termination. In such cases the hypotension is of some value as a sign of the impending end, but usually it is but one of many. As a rule, the terminal fall in pressure may be measured by minutes or hours, not by days.

B. Collapse and Shock.—a. General Features.—There are two conditions, the one medical, the other surgical, in which hypotension is more than a symptom; it is the essential manifestation of the serious disorder of function which lies back of the clinical picture, and it is the chief source of danger to life. These two conditions are called collapse and shock. Collapse is seen in acute disease at the height of the infection, and, when severe, results rapidly in a fatal issue. It follows large hæmorrhages, but in this form will not be discussed here, since the causative factor, loss of blood-volume, is somewhat different. It also makes an appearance under the same circumstances which lead to shock, especially prolonged operations, in particular those involving repeated manipulation of the intestines and insults to sensory nerve trunks, or accompanied by profuse hæmorrhage. Though there is much discussion alike over the definition and the causation of shock, there seems no good reason for considering it more than the fully-developed condition, of which collapse is the forerunner. Both shock and the collapse of the acute infections have, in the last few years, been the subject of much fascinating experimental study. The former has aroused widespread interest among surgeons, and will be considered in detail in the chapter devoted to blood-pressure in surgical conditions. The latter, unfortunately, in view of the importance of the work done, has attracted much less attention than it should from physicians and pathologists in our country.

Sudden death during the height of an acute infection, be it pneumonia or diphtheria, or, as is less usual, typhoid fever, nine times out of ten is set down on the death certificate, and in more pretentious medical literature, as "heart failure." However, the phenomena often resemble those of profuse hæmorrhage, and, in typhoid fever especially, the differential diagnosis between the two offers great practical difficulty. The pulse in collapse becomes small, empty, and rapid; there is no systemic venous congestion or pulmonary œdema; merely extreme prostration, cold skin, the changed facies, and the ineffectual heart. This striking similarity to death from hæmorrhage, and the absence of the usual sequences of cardiac asystole, have made numerous clinicians interrogate the ordinary interpretation and ask themselves whether, after all, the patient's vaso-motor mechanism might not have failed; thus, as it were, bleeding him to death into his own veins (see page 18).

b. Experimental Evidence.—This question has been approached from the experimental side during the last five years, with results which seem to me quite conclusive. The first paper of Romberg and Pässler should be read by every one who cares to see purely clinical problems elucidated by the methods of experimental physiology. They studied the fatal collapse which occurred in rabbits after infection with the pneumococcus, the bacillus pyocyaneus, and the diphtheria bacillus; the first producing a true septicæmia, the latter a local lesion with general toxæmia. All of the two hundred and fifty animals used were autopsied, and the heart and other important organs examined microscopically. Their method consisted in observing the mean carotid pressure at different stages of the disease, and the effect upon it of (1) abdominal massage, which increased the work of the heart, by supplying it with more blood; (2) compression of the aorta above the diaphragm, which makes the work of the heart maximal; (3) irritation of the nasal mucous membrane with a Faradic current, which causes extreme reflex vaso-constriction; and (4) short asphyxia (30 secs.), which acts similarly, only on both medullary and spinal vaso-motor centres, while sensory stimulation affects only the centre in the medulla. They reasoned that, should there be no rise in pressure from sensory irritation or suffocation, while abdominal massage and ligature of the aorta still called forth a well-marked one, then the heart must be functionally capable and the vaso-motor mechanism paralyzed. To determine whether the central or peripheral vaso-motor mechanism was at fault, they used injections of barium chloride, which cause constriction of the arteries by purely local action upon them.

Their experiments showed that the blood-pressure and the response to all the procedures remained perfectly normal throughout the early stage of the disease, being unaffected by the fever. The greatest elevation of pressure was obtained on stimulating the mucous membrane of the nose. When the animals showed signs of impending collapse in their behavior, the blood-pressure, though still normal, began to sink, while the heart beat more forcibly. Hand in hand with this went a great reduction in the rise of pressure from sensory irritation, a moderate decrease in the asphyxial elevation, but as high a pressure as before after abdominal massage. In many cases the pressure did not fall until the reflex rise had been almost abolished, evidently being maintained by increased cardiac energy, in spite of the vascular dilatation. Finally, in complete collapse, which developed very rapidly, the aortic pressure fell to the lowest level, as after destruction of the spinal cord; no reflex rise could be obtained, but abdominal massage gave an immediate elevation. It was evident, therefore, that the circulatory disturbance at the height of the infection depended absolutely upon a paralysis of the vessels, not upon any damage to the force of the heart.

As regards their reaction to compression of the thoracic aorta, the diphtheria animals showed a divergence from the pneumococcus ones; the latter evincing practically normal cardiac reserve force, while the former showed a distinct falling off. Anatomically, also, the diphtheria hearts had suffered damage, parenchymatous degeneration being well marked, as in clinical diphtheria. The pneumococcus animals had scarcely any change in their cardiac muscle. This evidence of weakness in the heart muscle in diphtheria was of minor importance, the real cause of death in all cases being the complete loss of vaso-motor tone. By intravenous injections of barium chloride they proved decisively that this was due to central paralysis. Their conclusions were, that all three organisms used damage the circulation through paralyzing the vaso-motor centres throughout the medulla and cord; this vaso-motor paralysis leads to a fall in blood-pressure, and further, to a changed blood distribution; the splanchnic circulation is overfilled, the brain, muscle, and skin vessels are empty; the heart is not affected, except secondarily through insufficient blood-supply. These conclusions applied only to the deaths at the height of the infection, not to those at a later stage in the disease, or during convalescence. Another piece of work from the Leipzig clinic, by Hollwachs, published at the same time, brought these later deaths, in diphtheria, into relation with the graver structural changes in the heart (acute infectious myocarditis).

In line with this investigation, Heineke showed later that a paralysis of the medullary centres is the cause of death in perforative peritonitis, a condition almost invariably associated with profound collapse in man. He drew attention to the fact that the heart continued to beat after respiration had ceased. Hasenfeld and Fenyvessy also demonstrated the slight functional damage to the heart in acute fatty degeneration caused by phosphorus, and considered the cause of death in artificial phosphorus poisoning a vascular paresis; a position which Pal justifies from clinical observations.

Hasenfeld and Fenyvessy, as well as Romberg and Pässler, found some loss of power in the fatty hearts, nevertheless. Since that time, the cardiac factor in collapse during diphtheria has received special attention. Rolly, by carefully planned experiments with diphtheria toxin, injected into an isolated heart-lung circulation (Hering's method), found a direct action of the poison upon the heart, which led rapidly to a fatal issue in spite of artificial respiration. Most interesting in his experiments was the length of the latent period, often as much as twenty-four hours, during which no symptoms occurred after the most enormous doses of toxin. When the heart began to fail, death followed rapidly, and nothing could ward it off; perfusion of the heart with normal blood being insufficient to wash out the poison. Rolly predicated a gradual fixation of the toxin in the cardiac muscle as the explanation of these phenomena. Of late v. Stejskal has combatted the theory of vaso-motor death in diphtheria, and, in elaborate experiments, in which he measures aortic and left auricle pressures simultaneously, endeavors to show that failure of the heart is, after all, the primary cause of the fatal hypotension. His method is not wholly convincing, and he admits the involvement of respiratory and vaso-motor centres as well. Pässler and Rolly have met his criticisms and, by a more thorough application of his own method, they have shown that, while damage to the heart is actual, nevertheless it is not the cause of death at the acme of the infection. This, in diphtheria, as in pneumococcus septicaemia, is due to vaso-motor paralysis, which the heart is able for a time to counteract.

For surgical shock, Crile's exhaustive experiments seem to give us an equally secure basis for regarding the clinical picture as one of extreme and dangerous hypotension, due to exhaustion of the central vaso-motor mechanism. He approached the problem by similar methods, and his book, "Blood-Pressure in Surgery," should be read by all who desire the evidence at first hand. His experiments numbered two hundred and fifty-one, on two hundred and forty-three animals. All were reduced to full surgical anæsthesia. Shock was induced, as a rule, by exposure and manipulation of the intestines, or by skinning the animal. The blood-pressure fell progressively, and the reflex rise from stimulation of the sciatic nerve, or peripheral irritation (burning the paw), diminished pari passu with it. When the animal was in profound shock, which required at least half an hour for its development, the blood-pressure was about 20 mm., practically no reflex rise could be obtained, and strychnin was powerless to affect it. The sequence of events was identical in animals whose hearts had previously been isolated from the central nervous system.

In the most profound shock it was possible to raise the blood-pressure to almost any level, and maintain it there for long periods of time, by the continuous intravenous infusion of adrenalin in salt solution, 1 in 50,000 to 100,000. A decapitated dog was kept alive ten and a half hours in this way. By increasing the surrounding atmospheric pressure, in a pneumatic chamber or tube, the arterial pressure could be controlled at will, and paralleled the movements of external pressure. This prompt response of the blood-pressure, when sufficient peripheral resistance was supplied, proved conclusively that the heart was not at fault; just as in Romberg and Pässler's experiments, ligature of the aorta demonstrated the functional capacity of the heart in the collapse of acute disease. On the other hand, the complete abolition of the reflex rise in pressure, and of a rise from central stimulation with strychnin, while adrenalin produced normal vaso-constriction, showed that central vaso-motor paralysis was the real cause of the hypotension.

Porter and Quinby have recently interposed some objections to the theory that exhaustion of the vaso-constrictor centre, or neurons, is the essential cause of shock. These rest upon a few experiments, in which they proved that stimulation of the central end of the depressor nerve produced the same percentage fall in blood-pressure, in rabbits and cats, after shock had been induced, as in their normal state. As one of their protocols shows an identical result after section of the dorsal cord, which eliminates the medullary centre entirely, I am unable to follow their reasoning. I cannot see that they have eliminated a vagus depression of the heart, which Crile found was not at all diminished in shock. Neither do they indicate the depth of shock produced.

A rapid fall in blood-pressure during operation, due to hæmorrhage or other causes, in which the vaso-motor centre has not become exhausted and will still respond to peripheral or central stimuli, is called collapse. Between it and shock, Crile draws a sharp line. I am unable to see that the distinction is more than one of degree, but it unquestionably justifies Crile's methods of treatment in the profound cases. This will be discussed in more detail in the chapter relating to blood-pressure in surgical conditions.

c. Practical Results.—In the light of all this evidence, I think we must regard collapse and shock, whether seen medically or surgically, primarily as disturbances of the functions of the vaso-motor centres. Both for prognosis, and as a guide to treatment, therefore, the sphygmomanometer becomes valuable. It alone can give warning of the onset of the hypotension, which accompanies vaso-motor paralysis. Crile's mechanical support of the circulation by a pneumatic suit is the direct outcome of his blood-pressure researches, and promises much in the treatment of operative shock. In acute disease, far too little use has been made of blood-pressure observations to control treatment. The only careful work along these lines has been that of Cook, and of Cook and Briggs. Their results are so encouraging that blood-pressure charts should become common, at least in hospitals. In private practice I have found that an intelligent nurse can easily make the necessary systematic observations on these cases.

C. Effects and Dangers of Hypotension.—Abnormally low blood-pressure must lead to an accumulation of blood in the veins and a slowing of the current in the arteries, if it be progressive. This will of course affect the capillary circulation, and the nutritive and secretory processes dependent on it. The great accumulation of blood in the abdominal veins, which results from extreme loss of vaso-motor tone, naturally affects the blood distribution markedly, and lessens the amount of heat given off from the surface of the body, thus increasing fever. Most serious, however, is the danger to the heart. Both in physiological experiment (see page 18), and in pathological, we have found that complete loss of vaso-motor tone soon leads to death, because the ventricles discharge their contents into the flaccid arteries, and receive less and less blood from the relaxed veins. The diminished energy of contraction with diminished ventricular contents, which Frank demonstrated (see page 15), completes the vicious circle. Pal's observations of the empty ventricles, found at autopsy in persons dying of phosphorus poisoning, are the pathological evidence of this mode of death.

4. THE RELATION OF BLOOD-PRESSURE TO PULSE-RATE AND TEMPERATURE

Marey used to teach that arterial pressure and pulse-rate varied inversely to one another and, in fever especially, that higher temperature and more rapid pulse went hand in hand with diminished pressure. His reasons for this were in part sphygmographic, in part theoretical. The older clinical statements, from the evidence of the less accurate instruments, were usually positively for or against Marey's teaching. Wetzel, and Reichmann believed a fall to be the rule in fever, Zadek, and Arnheim taking the contrary view and claiming a rise. Giglioli thinks the relation is usually an inverse one. Mosen, and Kuhe-Wiegand, however, among the older writers, and more recently Alezais and Francois, Hensen, and many others, have demonstrated that the real reason for such mutually conflicting opinions lies in the absence of any definite relation between the blood-pressure and the temperature. Potain looks on the infection as the cause of the lowering of pressure usual throughout acute febrile diseases. He sees in the fever itself an influence for elevation, which makes itself felt in the lesser rises and falls from hour to hour.

As a matter of fact, the blood-pressure in febrile disease is subject to many of the causes for variation which exist during health, in addition to many more. In consequence, no attempt to explain the smaller features of the pressure curve, under such circumstances, can hope to be successful. The findings of Romberg and Pässler, in the early stage of experimental infections, show that fever, of itself, is inefficient to alter mean blood-pressure. That says nothing as to possible changes in the relation of systolic and diastolic pressures to one another. It is to be hoped that a study of this in fever, as in other conditions, may give us new insight into the circulatory changes which are certainly associated with the temperature changes. The dicrotic pulse, for instance, may find more satisfactory elucidation in this way. I have evidence that it is not necessarily associated with low systolic pressure. In one case the pulse-pressure variation was abnormally increased, as would be expected.

5. BLOOD-PRESSURE AS A GUIDE TO THE FUNCTIONAL ABILITY OF THE HEART AND THE ADEQUACY OF THE CIRCULATION

Strangely though it sounds, the mean blood-pressure alone tells us absolutely nothing of the heart's ability to carry on the circulation. Clinically, normal and even somewhat high tension may be observed in patients dying of slow cardiac failure. On the other hand, animal experiments have shown that fatal hypotension occurs with a heart abundantly able to maintain a high-pressure, if it were supplied with a sufficient peripheral resistance. The explanation is simple, when one remembers the reciprocal relations of heart and vaso-motor system. A very small amount of blood may enter the aorta at each systole, but if the arterioles be so tightly contracted that only a similar amount can escape into the capillaries, the aortic pressure will be unchanged. In such a case the systolic and diastolic variation will be slight. On the other hand, even though the heart deliver five times the normal output in a minute, if the arteries are so dilated that six times the usual quantity passes on into capillaries and veins, the aortic pressure will rapidly fall. Here the pulse-pressure change will be large, the diastolic pressure very low, and the systolic perhaps normal. It is therefore evident that the measurement of the two pressures will give us much more information about the actual state of the circulation than either alone, or than the mean pressure, because affording some little insight into the systolic pulse-volume of the left ventricle. Erlanger and Hooker believe that, under perfectly normal conditions, the pulse-pressure (difference between diastolic and systolic pressure) varies directly with the acceleration of blood-flow produced by each pulse-wave.

In pathological conditions there are too many possible variables to justify such an opinion. When the distensibility of the arterial walls is diminished by disease, or more especially by high internal tension (see page 25), a small amount of blood injected into the aorta will raise systolic pressure far more than an equal amount at lower pressure. This does not at all indicate a corresponding augmentation of blood-flow. As a matter of fact, though changes in the work of the heart through variations in aortic pressure are marked, the effect of output variations greatly outweighs them. Zunst has shown that the heart is in a position to multiply its work fivefold in an instant, to supply the blood needed for ordinary muscular activity. The possible maximum volume increase is much larger. In comparison with such figures, even a doubling of mean aortic pressure looks trifling. Therefore, with the chief factor in the heart's work unmeasurable, we should be chary of our deductions from a known factor of secondary importance.

Here, as everywhere in medicine, we must correlate all the evidence, of which the reading of the sphygmomanometer is but part. A heart may be carrying on the circulation normally in bed, but be inadequate to supply the demands of active life. Blood-pressure determinations during exertion, as made by Moritz, might give light on this, but the subjective evidence of dyspnœa and precordial distress are more readily at hand. Again, with hypotension, the heart may be maintaining the circulation with all its reserve force, or with only its ordinary expenditure of energy; no measurements can tell us which.

We may at least feel sure that, with normal or high pressure, the absence of dyspnœa or distress on exertion argues perfect cardiac function at the time; also that, with low pressure, the clinical evidence of a disturbed circulation may coexist with a sound heart. In either case we obtain more conclusive evidence concerning the peripheral resistance. This has more than diagnostic and prognostic value. It may guide us to recognizing the necessity for attacking the circulation through the vaso-motor system, when this is at fault, and thus protect the heart from secondary damage, and needless or harmful stimulation.

LITERATURE

Adami, J. G. Notes upon Cardiac Hypertrophy. Montreal Med. Jour., 1895, vol. xxiii, p. 811.

Alezais et Francois. La tension arterielle dans la fievre typhoide. Rev. de Méd., 1899, vol. xix, p. 88.

Arnhein, Fr. Ueber das Verhalten des Wärmeverlustes, der Hautperspiration und des Blutdrucks bei verschiedenen fieberhaften Krankheiten. Zeitschr. f. klin. Med., 1882, vol. v, p. 363.

Askanazy, S. Ueber den Wassergehalt des Blutes bei Kreislaufstörungen, Nephritiden, Anämien und Fieber, etc. Deutsch. Arch. f. klin. Med., 1897, vol. lix, p. 385.

v. Bamberger, H. Ueber Morbus Brightii und seine Beziehungen zu anderen Krankheiten. Volkmann's Sammlung klin. Vorträge, 1879, vol. clxxiii, p. 1552.

v. Basch, S. Die Herzkrankheiten bei Arteriosclerose. Berlin, 1901.

Bright, R. Cases and Observations Illustrative of Renal Disease Accompanied with the Secretion of Albuminous Urine. Guy's Hosp. Reports, 1836, vol. i, p. 335 (see p. 396).

Cohnheim, J. Allgem. Pathol., 1880, pp. 320–365.

——Ueber acutes Lungenœdem. Gesam. Abhandlungen, Berlin, 1885, p. 593.

Cook, H. W. The Clinical Value of Blood-pressure Determinations as a Guide to Stimulation in Sick Children. Am. Jour. of the Med. Sci., 1903, vol. cxxv, p. 433.

Cook, H. W., and Briggs, J. B. Clinical Observations on Blood-pressure. Johns Hopkins Hosp. Rep., 1903, vol. xi, p. 451.

Cushing, Harvey. Some Experimental and Clinical Observations Concerning States of Increased Intracranial Tension. The Mütterer Lecture for 1901. Am. Jour. of the Med. Sci., 1902, cxxiv, p. 375.

—— The Blood-pressure Reaction of Acute Cerebral Compression, Illustrated by Cases of Intracranial Hæmorrhage. Am. Jour. of the Med. Sci., 1903, vol. cxxv, p. 1017.

Crile, G. W. Blood-pressure in Surgery. Philadelphia, 1903.

Erlanger, J., and Hooker, D. R. The Relation between Blood-pressure, Pulse-pressure and the Velocity of Blood-flow in Man. Am. Jour. of Physiol., 1904, vol. x; Proceed. of Am. Physiol. Soc., p. xv.

Giglioli, G. Y. Alcune critiche e alcune richerche di sfigmomanometria clinica. Riv. crit. di clin. med., 1900, vol. i, pp. 625 and 641.

Gull, W. W., and Sutton, H. G. On the Pathology of the Morbid State commonly called Chronic Bright's Disease with Contracted Kidney. Medico-Chirurg. Transactions, 1872, vol. lv, p. 273.

Hasenfeld, Arthur. Ueber die Herzhypertrophie bei Arteriosclerose nebst Bemerkungen über die Herzhypertrophie bei Schrumpfniere. Deutsch. Arch. f. klin. Med., 1897, vol. lix, p. 193.

Hasenfeld und Fenyvessy. Ueber die Leistungsfähigkeit des fettig entarteten Herzens. Berl. klin. Wochenschr., 1899, vol. xxxvi, pp. 80 and 125.

Hasenfeld und Romberg. Ueber die Reservekraft des hypertrophischen Herzmuskels, u. s. w. Arch. f. exper. Pathol. u. Pharmakol., 1897, vol. xxxix, p. 331.

Heineke, H. Experimentelle Untersuchungen über die Todesursache bei Perforationsperitonitis. Deutsch. Arch. f. klin. Med., 1901, vol. lxix, p. 429.

Hensen, H. Beiträge zur Physiologie und Pathologie des Blutdrucks. Deutsch. Arch. f. klin. Med., 1900, vol. lxvii, p. 438.

Hill, Leonard. The Physiology and Pathology of the Cerebral Circulalation. London, 1896, pp. 149 and following.

Hirsch, Carl. Ueber die Beziehungen zwischen dem Herzmuskel und der Körpermuskulatur und über sein Verhalten bei Herzhypertrophie. Deutsch. Arch. f. klin. Med., 1900, vol. lxviii, p. 55.

Hirsch, C., und Beck, Carl. Studien zur Lehre von der Viscosität (inneren Reibung) des lebenden menschlichen Blutes. Deutsch. Arch. f. klin. Med., 1902, vol. lxxii, p. 560.

Huchard, H. Traité cliniques des maladies du cœur et des vaisseaux. Paris, 1893.

Israel, O. Experimentelle Untersuchung über den Zusammenhang zwischen Nierenkrankheiten und secundären Veränderungen des Circulationssystems. Virchow's Archiv, 1881, vol. lxxxvi, p. 299.

John, Max. Ueber den arteriellen Blutdruck der Phthisiker. Zeitschr. f. diät. u. phys. Therap., 1902, vol. v, p. 275.

Johnson, Sir George. The Pathology of the Contracted Granular Kidney. London, 1896.

Kapsammer, Geo. Blutdruckmessungen mit dem Gärtner'schen Tonometer. Wien. klin. Wochenschr., 1899, vol. xii, p. 1279.

Kocher, Th. Hirnerschütterung, Hirndruck, etc. Nothnagel's spec. Pathol. u. Therap., vol. ix, III. Th., II. Abth., Wien, 1901.

Krehl, L. Pathologische Physiologie. Leipzig, 1898.

Die Erkrankungen des Herzmuskels und die nervösen Herzkrankungen. Nothnagel's spec. Pathol. u. Therap., vol. xv, I. Th., V. Abth., Wien, 1901.

Kuhe-Wiegand, H. Ueber den Einfluss des Fiebers auf den arteriellen Blutdruck. Arch. f. exp. Pathol. u. Pharmakol., 1886, vol. xx, p. 126.

Lewy, Benno. Die Arbeit des gesunden und des kranken Herz. Zeitschr. f. klin. Med., 1897, vol. xxxi, pp. 321 and 520.

Marey, E. J. La circulation du sang a l'état physiologique et dans la maladies. Paris, 1881, pp. 334 and 564.

Moritz, O. Der Blutdruck bei Körperarbeit gesunder und Herzkranker Individuen. Deutsch. Arch. f. klin. Med., 1903, vol. lxxvii, p. 339.

Mosen, R. Ueber das Verhalten des Blutdrucks in Fieber. Deutsch. Arch. f. klin. Med., 1894, vol. lii, p. 600.

Pal, I. Ueber Gefässtod. Wien. klin. Rundschau, 1899, vol. xiii, p. 229.

Pässler und Rolly. Experimentelle Untersuchungen über Kreislaufsstörungen bei acuten Infectionskrankheiten. Deutsch. Arch. f. klin. Med., 1903, vol. lxxvii, p. 96.

Porter, W. T., and Quinby, W. C. The Condition of the Vaso-motor Neurons in "Shock." Boston Med. and Surg. Jour., 1903, vol. cxlix, p. 455.

—— The Condition of the Vaso-constrictor Neurons in "Shock." Am. Jour. of Physiol., 1904, vol. x. Proceed. of Am. Physiol. Soc., p. xii.

Reichmann, Ed. Ueber das Verhalten des arteriellen Blutdruckes in Fieber. Deutsch. med. Wochenschr., 1889, vol. xv, p. 784.

Riegel, Fr. Ueber die Veränderungen des Herzens und des Gefässsystems bei acuter Nephritis. Zeitschr. f. klin. Med., 1884, vol. vii, p. 260.

Rolly, F. Ueber die Wirkung des Diphtheriegiftes auf das Herz. Arch. f. exp. Pathol. u. Pharmakol., 1899, vol. xlii, p. 283.

Romberg, E., und Pässler, H. Experimentelle Untersuchungen über die allgemeine Pathologie und Therapie der Kreislaufsstörung bei acuten Infectionskrankheiten. Deutsch. Arch. f. klin. Med., 1899, vol. lxiv, p. 652.

Rosenbach, O. Die Krankheiten des Herzens. Wien, 1897, pp. 551–579 and 742.

Sansom, A. E. The Diagnosis of Diseases of the Heart and Thoracic Aorta. Philadelphia, 1892.

Senator, H. Die Erkrankungen der Nieren. Wien, 1902, pp. 110–129.

Sibson, F. The Influence of Bright's Disease: (1) On the Heart and Arteries: (2) On the Production of Inflammation. Lancet, 1874, vol. i, pp. 437 and 505.

v. Stejskal, K. Kritisch-experimentelle Untersuchungen über den Herztod in Folge von Diphtherietoxin. Zeitschr. f. klin. Med., 1902, vol. xliv, p. 367; and 1903, vol. li, p. 129.

Sumikawa, P. Ein Beitrag zur Genese der Arteriosklerose. Beitr. zur pathol. Anat. u. zur allgem. Pathol., 1903, vol. xxxiv, p. 242.

Traube, L. Gesam. Abhandlungen. Berlin, 1856, vol. ii, p. 290; vol. iii, p. 440.

Wetzel, A. Ueber den Blutdruck im Fieber. Zeitschr. f. klin. Med., 1882, vol. v, p. 323.

Zunst, N. Die Ernährung des Herzens und ihre Beziehung zu seiner Arbeitsleistung. Deutsch. med. Wochenschr., 1892, vol. xviii, p. 109.

CHAPTER VII

BLOOD-PRESSURE IN INTERNAL DISEASES

1. Diseases of the heart, arteries, and kidneys.

A. Nephritis.

- a. Chronic interstitial nephritis.
- b. Other forms of chronic nephritis.
- c. Acute nephritis.
- d. Uræmia.

B. Cyclical albuminuria.

C. Arterial disease.

- a. General.
- b. Of the cerebral vessels. Apoplexy.
- c. Other local processes. Thrombosis; aneurism; compression

D. Diseases of the heart-muscle.

E. Valvular heart-lesions.

- a. Aortic insufficiency.
- b. Other valve-lesions.
- c. Acute endocarditis.

F. Neuroses of the heart.

- a. Exophthalmic goitre.
- b. Other neuroses.

G. Prominent cardiac symptoms.

- a. Angina pectoris.
- b. Cardiac asthma and other forms of dyspnœa.
- c. The arrhythmias.

H. Effects of exertion on blood-pressure in cardio-vascular disease.

I. Therapeutics.

Literature.

2. Acute infectious diseases.

- A. Typhoid fever.
  - a. Hæmorrhage and collapse.
  - b. Perforation.
  - c. Therapeutics.
  - d. Late effects.
- B. Pneumonia.
- C. Diphtheria.
- D. Other acute infections.
  - Malaria; acute rheumatism; septic conditions; influenza.
- E. Acute infections in childhood.
  - a. Therapeutics.

3. Chronic infectious diseases.

- A. Tuberculosis.
  - a. Hæmoptysis.
- B. Syphilis.

4. Chronic diseases associated with impaired nutrition.

- A. Diabetes.
- B. The anæmias.
- C. Cachectic states.
  - a. Addison's disease.

5. Miscellaneous.

- A. Lead poisoning.
- B. Gout.
- C. Emphysema. Chronic bronchitis. Asthma.
- D. Pleural and peritoneal effusions.
- E. Hæmorrhage.

Literature.

CHAPTER VII

BLOOD-PRESSURE IN INTERNAL DISEASES

1. DISEASES OF HEART, ARTERIES, AND KIDNEYS

THE tabulation of the results of blood-pressure observations, as of all other medical statistics, is most unsatisfactory in those conditions which least readily lend themselves to a uniform classification, and in which the personal equation of the observer most conspicuously enters into the diagnosis. Acute diseases are, in the main, easily recognized, and are always placed in the same category. Chronic diseases, however, especially those which involve the circulatory system and kidneys, are difficult of analysis, even at the post-mortem. In most instances heart and arteries are both involved in chronic disease of the kidney, and, vice versa, many primary cardiac lesions lead in time to renal involvement. Since the proper classification of these cases offers such difficulties to the pathologist, what wonder that their clinical diagnosis should be so largely a matter of individual opinion. I shall therefore at the outset ask pardon for giving my own observations an apparently undue weight in this section, because they are the only ones whose inherent error I can at all calculate.

A. Nephritis.—a. Chronic Interstitial Nephritis.—The clinical picture of "the genuine contracted kidney" of the Germans, "the small granular kidney" of the English writers, is in most cases sufficiently clear-cut to make the diagnosis during life secure. It is essentially a disease of kidneys, arteries, and heart combined. Permanent high blood-pressure is one of its salient features, higher than is usually seen in any other condition. Recent sphygmanometric observations have only emphasized this already well-known fact. A systolic pressure of more than 200 mm. (12 cm.), 240 mm. (5 cm.), is not uncommon. Diastolic pressure will be 60 to 80 mm. lower. I have seen a number of cases that gave readings over 300 mm. (5 cm.),

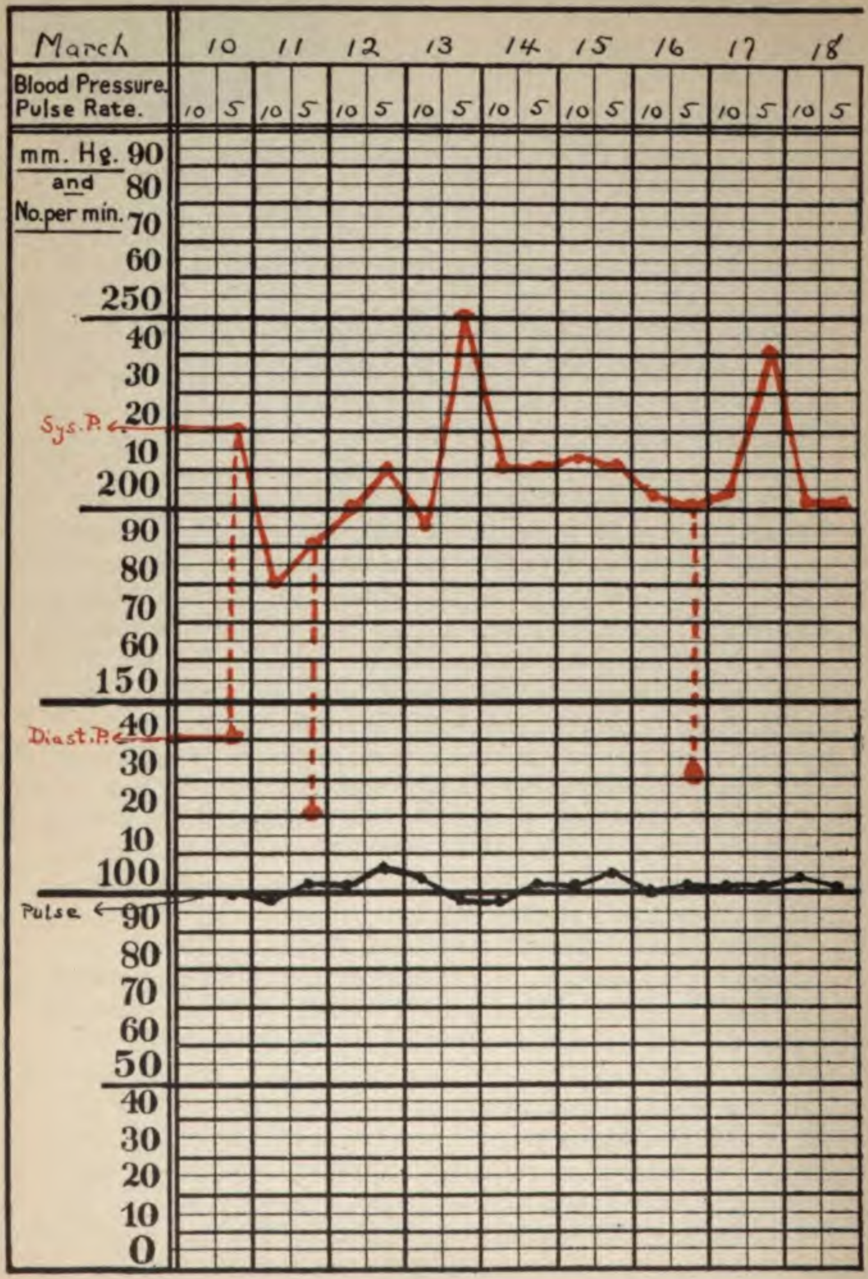

March
10
11
12
13
14
15
16
17
18
Blood Pressure.
Pulse Rate.
10
5
mm. Hg. 90
and 80
No. per min. 70
60
250
40
30
Sys. P.
20
10
200
90
80
70
60
150
Diast. P.
40
30
20
10
100
Pulse
90
80
70
60
50
40
30
20
10
0

FIG. 42.

FIG. 42.—MARKED HYPERTENSION IN CHRONIC INTERSTITIAL NEPHRITIS.
(Author's and Erlanger's sphygm. 12 cm.)

Chart of morning and evening systolic pressure and pulse-rate, with occasional measurement of diastolic pressure, from S. A., female, aged fifty-six, City Hospital.

March 10th; nose-bleed, 6 oz., for which large doses of nitroglycerin were given (see Fig. 52). Note the lower pressure next morning.

March 10th to 13th; taking nitroglycerin, gr. $^1/_{100}$, q. 1 h.

March 14th to 16th; hot pack, b. i. d.

March 17th and 18th; taking potassium iodide, gr. x, t. i. d.

Note the absence of therapeutic effect on the average level of blood-pressure.

Observations by Dr. W. C. Garvin.

but the error must have been large. The highest pressure in chronic nephritis that I have met with, since using the 12 cm. armlet, is 255 mm. systolic, 180 mm. diastolic.

Cases of contracted kidney without hypertension, though very rare, do occur. Senator calls attention to the fact that cardiac hypertrophy may fail when severe wasting disease is present. When seen at the very end of life, in hospital practice, the pressure may not be high, especially if the heart has become insufficient. The same cases earlier would probably have shown marked hypertension. Gross, however, reports a case in which small kidneys were found at autopsy, and which was under observation six weeks, with pressures between 120 and 145 mm. (R. R. 5 cm.), rising to only 160 mm. during a uræmic seizure. No other recent observers, of whom Buttermann, Carter, Cook and Briggs, Hensen, Gumprecht, Norris, and Potain give especial attention to nephritis, report an undoubted case of contracted kidney with continued low pressures. Hensen found very high readings even when advanced phthisis was also present.

Diagnostically, hypertension is of the first importance in connection with the contracted kidney. In my experience it is one of the most constant and, with the sphygmomanometer, most easily detected evidences of the disease. The enlargement of the heart requires skill to make out, and, with obesity, baffles the expert. The urinary changes are not constant. Albumin and casts are frequently absent, and even quantity and specific gravity may be normal at certain periods. A single examination in office practice may easily fail to reveal anything noticeably abnormal. The great advantage of routine blood-pressure measurements is, that hypertension will certainly be detected. While not a pathognomonic sign, it is, nevertheless, so striking that it puts the physician at once on the alert. Given a systolic pressure of over 200 mm., the diagnosis of contracted kidney must be disproved by repeated examinations before it is abandoned.

The most striking illustration of this in my recollection was the case of a priest of fifty-six, who consulted my father on account of three epileptiform convulsions which had come, without known cause, in the course of six months. It was impossible to prove cardiac hypertrophy. The urine showed a trace of albumin and a few hyaline casts, with perfectly normal quantity, specific gravity, and urea output. The question was between true epilepsy and uræmic convulsions. The blood-pressure, which was over 240 mm. (R. R. 5 cm.), threw the scale in favor of their uræmic nature. Subsequent examinations showed more constant urinary changes, and he died in less than a year.

Patients with chronic nephritis frequently consult a physician for nervous symptoms, for headaches, anginal pain on exertion, slight dyspnœa, or even severe cardiac asthma, and, because no murmurs are present and the hypertrophy is overlooked, they are assured that their hearts are normal. Such mistakes would be impossible were the sphygmomanometer in ordinary use. I saw the other day a lady of forty, who for two years had had considerable shortness of breath on exertion. At times she also suffered from attacks of nocturnal dyspnœa, with cough and raising of thin mucus, sometimes pink-tinged. She was not supposed to have any serious trouble. On account of her stoutness it was impossible to say anything about the heart, except that there were no valvular murmurs. The pulse tension did not feel extreme, but the sphygmomanometer showed systolic 255 mm., diastolic 180 mm. (J. 12 cm.); systolic 300 mm. + (R. R. 5 cm.). Urine examination later did not reveal marked change in quantity or specific gravity, but there was a fair amount of albumin, with hyaline, granular, waxy, and fatty casts. She was evidently having mild attacks of pulmonary œdema at night, with chronic nephritis and a left heart insufficient for the demands made on it by the extreme peripheral resistance.

Another patient, who had previously considered himself in perfect health, consulted a physician on account of slight disturbance of sensation and motion in right arm and leg, which came on suddenly with a transient aphasia. He had been twice examined when I saw him; nothing noted about his heart, and urine not looked at. He had marked hypertension and simple hypertrophy of the heart, with ringing second sounds. It was easy to predict that the urine would probably show where the primary disease lay, and the examination showed an enormous amount of albumin.

Such cases could be multiplied, but the lesson they have taught me is, that an instrument which so readily gives warning of the existence of high tension, which might otherwise be overlooked during hasty examinations, cannot be dispensed with. Our diagnostic methods are not yet so perfect that we can afford to pass by any reasonable addition to them.

In the primary contracted kidney I am unable to say that any definite relation exists between the actual height of blood-pressure and the expectation of life. One patient who, three years ago, had a reading above 300 mm. (R. R. 5 cm., to be sure on an enormous arm), is in as good condition to-day. A gradually increasing pressure, which resists treatment, is of bad

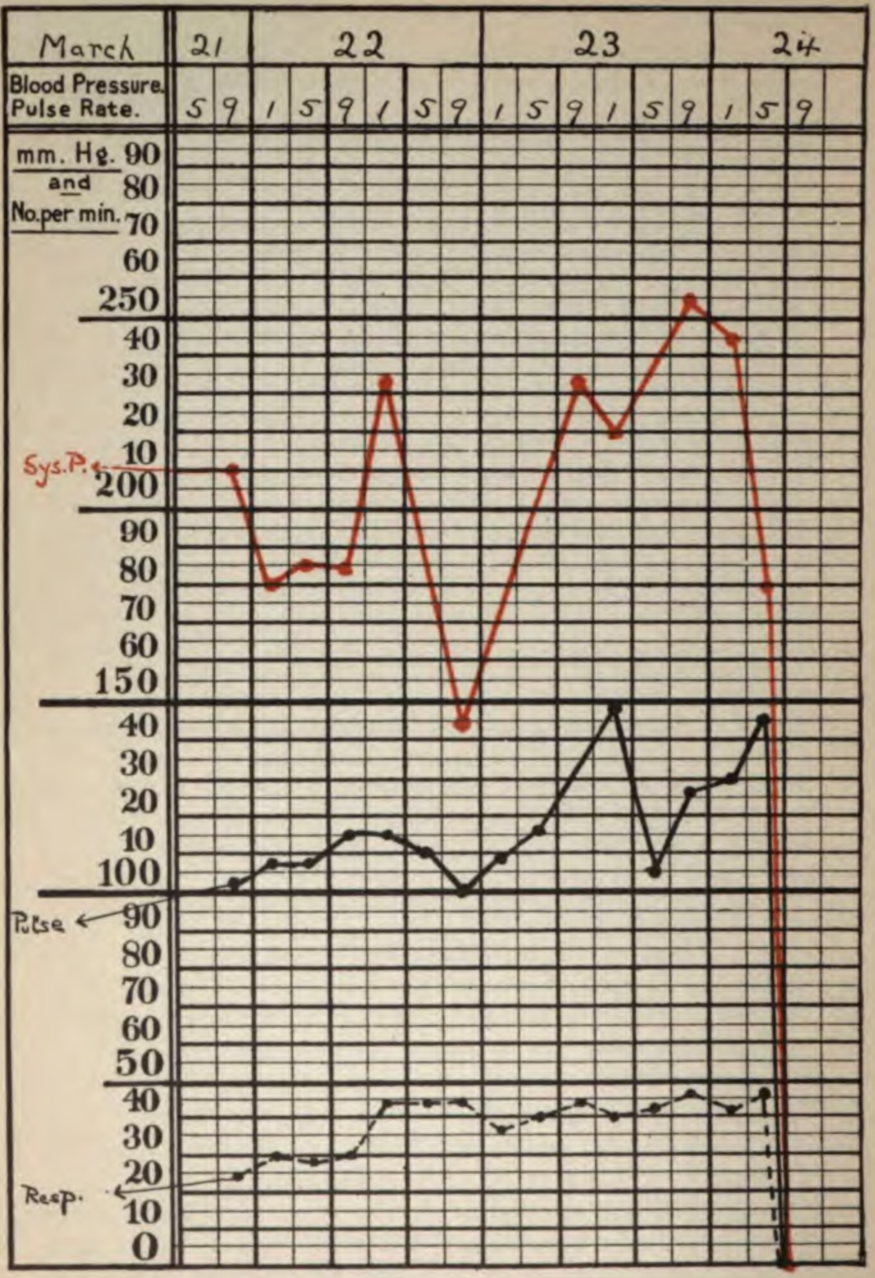

March
21
22
23
24
Blood Pressure.
Pulse Rate.
5 9 1 5 9 1 5 9 1 5 9 1 5 9 1 5 9
mm. Hg. 90
and 80
No. per min. 70
60
250
40
30
20
10
Sys. P.
200
90
80
70
60
150
40
30
20
10
100
Pulse
90
80
70
60
50
40
30
20
Resp.
10
0

FIG. 48.

FIG. 43.—CHRONIC NEPHRITIS; LEFT HEMIPLEGIA; LOBAR PNEUMONIA; DEATH.
(Author's sphygm. 12 cm.)

Chart from J. F., aged seventy-two, male, City Hospital. Old hemiplegia and chronic nephritis; pneumonia, right lower lobe, beginning March 20th, with high temperature range.

Autopsy showed gray hepatization of right lower lobe; marked cardiac hypertrophy (20 oz.); general and cerebral arterio-sclerosis; advanced chronic interstitial nephritis; areas of softening in each lenticular nucleus.

Note the maintenance of extreme hypertension until a few hours before death.

significance when there are any symptoms of cardiac insufficiency, because increasing the work of the already overtaxed left ventricle.

For instance, a gentleman of past fifty, with chronic interstitial nephritis and a much hypertrophied heart, had been subject to anginal attacks of moderate severity on exertion for several years. The pain came beneath the sternum, at times also down the left arm to wrist. He was formerly able to attend to his business and do ordinary slow walking without bringing on an attack oftener than every few weeks. In 1901 his systolic tension was 230 mm. (R. R. 5 cm.). In January, 1903, it was 270 mm., and it did not go below 265 mm. during the year; diastolic 190 to 200 mm. (Erl.). The attacks began to come after slighter exertion, in spite of the continued use of small doses of potassium iodide, which had always acted well with him formerly. By the latter part of December systolic pressure rose to 280 mm., and attacks were repeated several times daily. By taking nitroglycerin, gr. $\frac{1}{100}$ every three hours, he was much better for a time. This produced a fall of systolic pressure from 280 to 255 mm., of diastolic from 180 to 160 mm., in three minutes. Soon, however, the pain became worse again, and he was confined to his house, and was not seen again. He died in April.

Another feature in prognosis is the danger of cerebral apoplexy. Since it is impossible to predicate in any way the likelihood of local arterial disease in the brain, it must always be considered possible, and therefore extreme hypertension makes one fear rupture.

The following case, among many, shows such a termination, and, in addition, the history is of interest on account of the recurrence of severe attacks of œdema of the lungs, with comparative health between. The gentleman, forty-four years of age, had a sudden intense attack of pulmonary œdema at night, in July, 1900. This came without warning, lasted an hour, and was so severe that he lay on his face to let the serum, which he raised in great quantity, run out of his mouth. Between that time and May, 1902, he had three severe and five slighter attacks of a similar kind. Beyond slight dyspnœa on exertion, and occasional palpitation, he was well between. In May, 1902, he showed a heart coming half an inch beyond the nipple line, with booming first and much accentuated aortic second sound, and very slight aortic diastolic murmur. The arteries were somewhat diffusely thickened. Arterial pressure was 240 mm. (R. R. 5 cm., arm thin). The urine showed increased quantity and diminished specific gravity only. In June he had five attacks in seven days, after which he improved. Urine then showed a faint trace of albumin for the first time, but no casts. January 17, 1903, he became suddenly unconscious while in bed, showed complete left hemiplegia, and died in not much more than five minutes. At autopsy, Dr. E. H. Rogers found a large hæmorrhage in the right basal ganglia, but not breaking through into the ventricle. Such a termination has, of course, been well known since the days of Bright.

The sudden development of rising pressure, as we shall see later, may indicate the onset of acute uræmic manifestations, and deserves immediate attention. I believe the blood-pressure chart as essential for following cases of renal disease as is the temperature record in febrile patients. Figs. 42, 43, and 44 illustrate this.

b. Other Forms of Chronic Nephritis.—Here I purposely refrain from being more specific, because I do not believe the diagnosis of the various types of so-called chronic parenchymatous or diffuse nephritis can be made accurately during life. In none of them is high arterial pressure so constant as in the chronic interstitial form. In amyloid kidney, cardiac hypertrophy regularly fails, and Buttermann, Hensen, and Hayaski report cases with subnormal pressures. In the chronic hæmorrhagic form, normal or subnormal readings seem frequent. In typical chronic parenchymatous or diffuse nephritis, high tension may be as marked as in the contracted kidney.

One patient, a young woman of twenty-seven, has had abundant low-gravity urine, loaded with albumin and casts, for nearly five years, with some œdema of the legs and face at times. A year ago she began to show well-marked chronic uræmia; vomiting, weakness, breathlessness, and increasing œdema, and anæmia. She also developed albuminuric retinitis. Her systolic pressure, which was 260 mm. (R. R. 5 cm.) in January, increased to 270 mm. in March, and in June was 290 mm. She is still living, though in rather precarious condition. In all probability she is developing secondary contraction.

Another case, a man of twenty-five, had severe gonorrhœa with cystitis four years before. Two years later he developed headache and frequent urination, and, a few months later, œdema of face and pains in back. Nine months before he was seen he had both kidneys decapsulated by Edebohl's method, and, since operation, had suffered from constant headache, vomiting, and weakness, and had become very anæmic. He passed 65 to 80 oz. of urine in twenty-four hours, of about 1,010 specific gravity, containing very large amounts of albumin, and hyaline, granular, fatty, and epithelial casts. His heart was not enlarged to percussion, but the apex first sound was booming, and the aortic second sound was accentuated. Arterial pressure was 220 mm. (R. R. 5 cm.).

On the other hand, a man of fifty-three, who had shown albumin in the urine and œdema of the legs fifteen years before, and who for a year and a half had not been free from some œdema and albuminuria, without any heart lesion, showed, only two months before death, arterial pressure 120 to 130 mm. (R. R. 5 cm.).

Such a case cannot be classified, but shows the possibility of progressive fatal nephritis of long standing without high tension. In the milder forms of nephritis, cardio-vascular changes may not develop. Such a condition, for instance, is shown by a girl of thirteen, who had acute nephritis consecutive to diphtheria at three. She recovered at the time, but, for two years past, albumin, and hyaline, granular, and fatty casts had appeared in the urine frequently. Beyond a slight anæmia (hæmoglobin 75 per cent.), there were no clinical symptoms. Her systolic pressure was 105 mm., diastolic 85 mm. (Erl. 5 cm.). There could be no question of the existence of a chronic nephritis in spite of the low blood-pressure.

In the diagnosis of these forms of chronic nephritis the sphygmomanometer gives less aid. Hypertension, if present, is additional evidence, and points to the existence of cardio-vascular changes, with their attendant dangers. Its absence, however, in no way negatives the diagnosis. In connection with other findings, this negative evidence may be accorded some value, especially if the pressure be quite low. Such corroborative testimony, for instance, helps in deciding whether an albuminuria, which behaves in other regards like the cyclic form, can be considered of little import, or is an early sign of real inflammatory kidney disease.

So far as prognosis goes, as a rule the cases of nephritis, other than the chronic interstitial variety, have seemed more severe when associated with hypertension and cardio-vascular disease. This should be conditioned, however, as applying to cases in fair nutrition and able to be about. Where marked cardiac insufficiency or other complicating disease, such as phthisis, is present, low pressure may go hand in hand with the most serious outlook.

c. **Acute Nephritis.**—The term acute nephritis, as ordinarily met with, embraces an even more heterogeneous group of conditions than the last. Everything, from slight cloudy swelling to the most intense diffuse inflammation, may be included under it. One would scarcely expect the mild parenchymatous degenerations to influence blood-pressure. Even the more marked forms of secondary acute nephritis, which do not produce symptoms and which subside with the primary disease, that is, the nephritis which usually complicates the severe cases of typhoid fever and pneumonia, would not be likely to have hypertension when they give no other evidence of circulatory disturbance. Thus we find Carter stating that no case of this kind he examined showed high pressure. Buttermann, also, noted an absence of hypertension in mild acute nephritis, and in one case complicating typhoid, in which the urine showed much albumin, blood, and casts. Shaw says that acute kidney disease does not appear to cause increased pressure in children. I have recently seen a case of subacute nephritis, apparently syphilitic in origin, showing albuminuria, with epithelial, blood, waxy, granular, and hyaline casts, whose pressure was, systolic 105 mm., diastolic 85 mm. (J. 12 cm.).

On the other hand, Kaufmann and de Bary saw moderate hypertension in five cases of acute diffuse nephritis. Buttermann found quite high readings (210, 213, 193, 192 mm. R. R. 5 cm.) in a portion of his cases. One patient with acute scarlatinal nephritis had a 50 mm. rise within twenty-four hours from the onset of the kidney lesion. In his cases, the pressure fell with the disappearance of the clinical symptoms of renal disease. The older observations of Traube, Riegel, and others have already been alluded to. They make it clear that the nephritis following scarlet fever produces a rapid rise in arterial pressure. Sphygmomanometric observations on this form of acute Bright's disease are urgently needed, and should be recorded graphically in connection with the pulse, urine quantity, cardiac changes, and clinical symptoms. Such records, with autopsy observations and careful microscopic study of kidneys, splanchnic arterioles, and heart, would possess great value. They should be instituted in all contagious hospitals.

As to the so-called primary, or idiopathic acute nephritis, I can find no specific data. The diagnosis must be made with great caution. In hospitals, so far as my experience goes, an acute exacerbation of a chronic nephritis is the condition usually called primary acute nephritis. In such cases the hypertension would belong with the chronic process. I think it probable that the high pressure also shows an exacerbation during the acute symptoms.

The greatest difficulty in the way of satisfactory determinations during acute Bright's is the œdema. Even slight

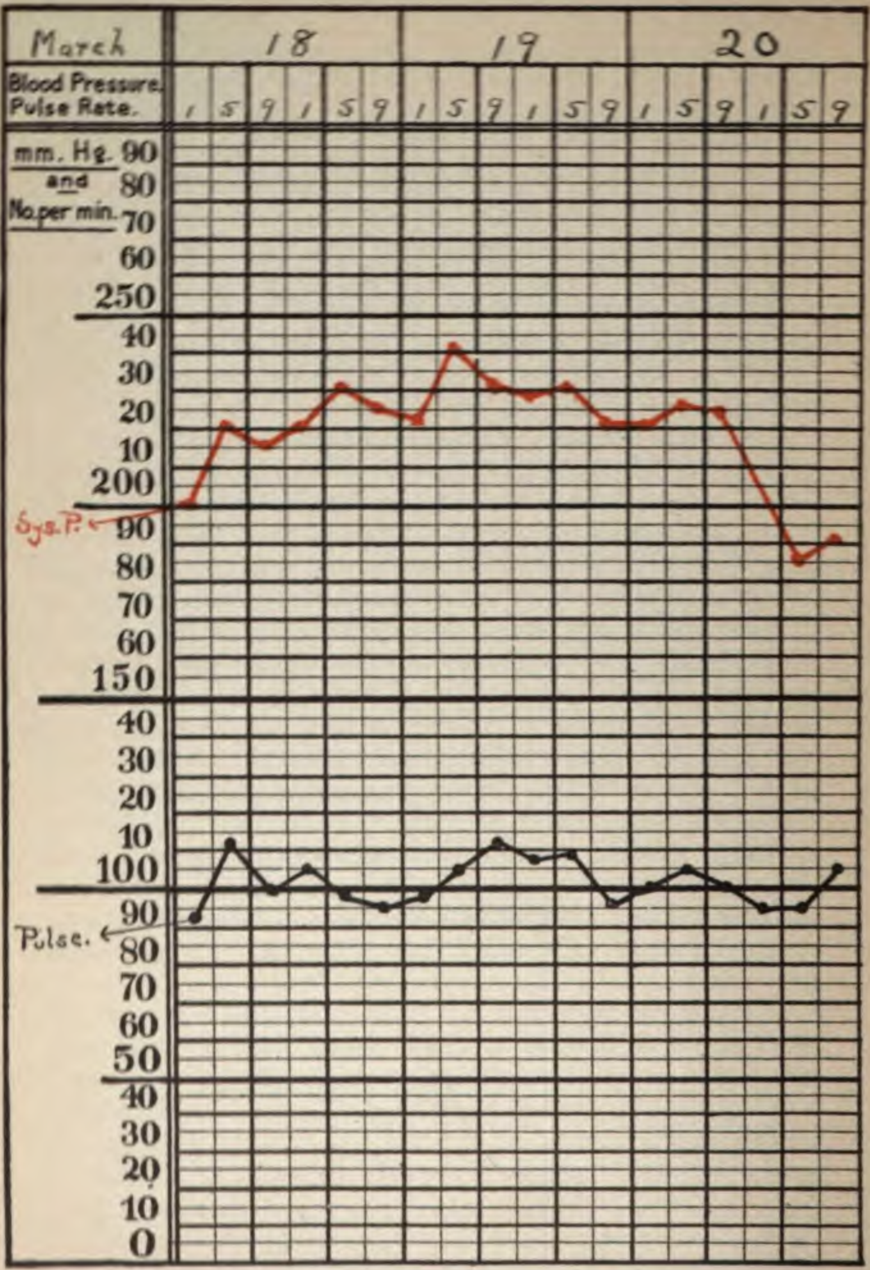

March
18
19
20
Blood Pressure.
Pulse Rate.
1 5 9 1 5 9 1 5 9 1 5 9 1 5 9 1 5 9
mm. Hg.
and
No. per min.
90
80
70
60
250
40
30
20
10
200
Sys. P.
90
80
70
60
150
40
30
20
10
100
90
Pulse.
80
70
60
50
40
30
20
10
0

FIG. 44.

FIG. 44.—CHRONIC URÆMIA. (Author's sphygm. 12 cm.)

Chart from J. M., aged forty-four, male, City Hospital. March 16th, began to have an exacerbation of a chronic nephritis, after an attack of acute gout; diminished urine, œdema of face, headache, vomiting, weakness. Took digitalis, saline diuretics, hot packs, and nitroglycerin, gr. $^{1}/_{50}$ q. 2. h., without effect.

March 19th, only nitroglycerin, gr. $^{1}/_{20}$ q. 2. h., with marked reduction of tension, disappearance of headache, and general improvement.

After March 21st tension rose again in spite of medication, falling ten days later with the advent of another acute gouty attack.

grades of it introduce an error, and, when distinctly appreciable, clinical blood-pressure measurements are impossible.

d. Uræmia.—It is difficult, in the literature of blood-pressure, to distinguish between acute and chronic uræmia. Most of the observations seem to bear upon the latter condition, but all are of real interest. There seems little question that the uræmic manifestations, that is, headache, vomiting, and disturbances of consciousness, run closely parallel with the blood-pressure. Fig. 44 is a clear example of this. Gross cites seven cases with continued observations, in which the symptoms increased with the tension and disappeared with its reduction through treatment, except that in the fatal cases a gradual fall in pressure ushered in the end. Cook and Briggs, in their Chart XVIII, show the former relation in a striking way, and, in Chart XXIX, fatal hypotension in collapse a few hours before death from uræmic coma. Laqueur first called attention to this important prognostic use of the sphygmomanometer. His cases showed a rise of 15 to 20 mm. (G.) every time the symptoms became worse, and a corresponding fall after packs, which had a beneficial result. The terminal fall in his patients began a few days before death, and was attendant upon obvious signs of heart weakness. Pal believes that headache and vertigo, transitory focal symptoms, amaurosis especially, and unconsciousness and coma, are alike dependent on high tension in the cerebral circulation. The first two, however, are frequently associated with conditions of cerebral anæmia, so the connection seems less obvious, though still perhaps circulatory. He is convinced of the great prognostic value of blood-pressure determinations when uræmia is feared.

Ascoli, in his book, believes that uræmic hypertension is an established fact, and cites a most interesting observation of Forlanini on an acute uræmia with convulsive seizures, which I think merits reproduction in detail here.

The patient, G. M., a man of forty-three, was admitted to the clinic November 30, 1896, with headache, dyspnœa, palpitation, and digestive disturbances, which had developed recently, and were increasing. He had had probable acute nephritis seven years before, cured in four months. He showed marked enlargement of the heart, with accentuated aortic second sound, but no murmurs. The pulse was frequent, small, and tense. Arterial pressure 250 to 270 mm. (R. R. 5 cm.). Urine, about 2,000 cc. in twenty-four hours, specific gravity 1,010 to 1,012, with moderate amounts of albumin and fairly normal total nitrogen. On a milk diet and potassium iodide the symptoms remained unaffected until January 17, 1897, when, after only slight dizziness and chilliness for a few hours, a severe convulsion occurred. The following table shows the progress of the case to the fatal issue five and a half hours later.

Time.	Blood-pressure, mm., Hg.	Pulse.	Remarks.
10.30	314	112	The uræmic attack has lasted a few minutes. Complete unconsciousness.
.32	320	. . .	Clonic convulsions, especially marked in right arm. Sweating. Narrow pupils.
.33	334	88	
.36	. . .	. . .	Venesection. 300 cc.
.39	294	86	
.41	280	80	
.42	. . .	. . .	Venesection. 300 cc. more.
.46	308	90	
.50	315	80	
.53	318	78	
.54	309	78	Vomiting.
11.03	294	62	Vomiting; continued unconsciousness.
.07	314	60	Sweating has ceased.
.16	316	63	Vomiting.
.28	318	60	
.33	330	57	
.35	330	60	Slight twitching in the right arm.
.43	. . .	. . .	Tonic spasm of the right arm.
.45	350	. . .	Clonic spasms of the upper limbs.
.46	348	. . .	General convulsion: involuntary micturition.
.54	. . .	128	Irregular pulse.
.57	350	128	The general convulsion continues.
12.02	. . .	. . .	Severe general convulsion.
.04	350	60	The convulsions are increasing in severity.
.09	. . .	. . .	The sphygmomanometric observation is discontinued. The patient died at 4 P. M., after a prolongation of the uræmic condition.

The autopsy revealed a great hypertrophy of the left heart; myocardium and valves healthy; hydropericardium, but no other dropsical effusion; small, fine-granular kidneys, with adherent capsule, yellow mottled surface, much contracted cortex, pale medullary substance. The anatomical diagnosis was contracted kidney with acute exacerbation, concentric cardiac hypertrophy, hydropericardium.

The highest tension I have met in uræmia, since using the 12 cm. cuff, was 290 mm., a day before death.

B. Cyclical Albuminuria.—The theory has been frequently advanced that the condition known as cyclical albuminuria, or physiological albuminuria, is dependent on altered circulatory mechanics in the kidney. The effect of posture in many cases is so striking that the name "orthostatic albuminuria" has been coined. Two recent studies with the sphygmomanometer lend their support to the hypothesis. Edel, in eight cases, found a fall in pressure under conditions which ordinarily cause a rise in the healthy man; viz., rising from bed, and cold baths. Coincidently with this fall, albumin appeared in the urine. In warm baths the primary rise in pressure was quickly succeeded by a fall, with albuminuria, and the same occurred after light exercise. He believes that his studies indicate the necessity for physical training and similar methods of treating the unstable circulation. Erlanger and Hooker made a careful study of the relation of blood-pressure and the pulse-pressures to the secretion of urine and albumin in one case. These pressure determinations with Erlanger's instrument are, of course, accurate. They found no constant relation between the amount of urine and albumin, and either systolic or diastolic pressure, or pulse-rate. They did prove, however, that the amount of urine increased and of albumin decreased with increase in the pressure variation of the pulse-wave. Since posture has a marked influence on this (see page 116), the pulse-pressure being greatest in the recumbent posture, the uniform absence of albuminuria in such cases during rest in bed, and its marked appearance after rising, are intelligible. Albumin excretion is known to be dependent on the rate of blood-flow through the kidney, the relation being an inverse one. These observations easily explain its variations in such cases, but not the reason why it should appear at all, when the normal man does not allow it to pass through his kidney parenchyma. Coupled with Edel's work they throw new light on a rather interesting condition, and are valuable chiefly as pointing the way toward the solution of some clinical problems by physiological methods.

C. Arterial Disease.—**a. General.**—The very striking relation between the distribution of the arterio-sclerotic process and the development of a hypertrophied left ventricle, which Hasenfeld and Hirsch demonstrated so clearly (see page 143), explains the sphygmomanometric findings in arterial disease. The clinical diagnosis of arterio-sclerosis usually means a palpable thickening of the radial, brachial, or temporal arteries. Sometimes even the sight of a normally tortuous, but abnormally exposed temporal, is considered adequate proof of disease. At the best we can but discover sclerosis, diffuse or nodular, with or without marked calcification, of the larger superficial arteries, by the sense of touch. In elderly persons without previous rheumatic history, the development of a systolic aortic murmur, or of a distinct lesion of the aortic valves, points toward atheromatous changes in the ascending aorta. Disease of the retinal vessels is visible through the ophthalmoscope, and from it we may infer similar changes in the cerebral arteries. Thus only a bare fraction of the arterial tree lies within reach of our methods of exploration, and the most important branches, those to the abdominal viscera, are entirely removed from approach. The sphygmomanometer is therefore a welcome adjunct to our other means of examination. In my experience and that of most others (Carter, Gumprecht, Hensen, Potain), a considerable degree of thickening of the superficial arteries may coexist with pressure not above the normal. In hospital practice, where poor nutrition and heart weakness complicate the conditions, even low normal pressures may be found. It is not alone the nodular form of arterio-sclerosis which fails to raise pressure. I have seen a case of diffuse thickening of the superficial arteries in a man not beyond middle life, where the reading was only 160 mm. (R. R. 5 cm.); and another with systolic 140 mm., diastolic about 115 mm. (J. 12 cm.). As a rule, however, the systolic pressure will usually be somewhat above normal, 160 to 180 mm. (R. R. 5 cm.), 145 to 160 mm. (12 cm.), and the diastolic very little raised, 110 to 130 mm. (5 cm.), 100 to 120 mm. (12 cm.); as, for instance, a man of seventy, with extreme calcification of brachials and radials, and systolic pressure 180 mm., diastolic 110 mm. (Erl. 5 cm.). Fig. 45 shows this moderate hypertension and its variations from day to day during an acute bronchitis.

On the other hand, in the cases of general arterio-sclerosis with marked hypertension, chronic Bright's cannot be eliminated without care. The combination is frequent, and gives the highest pressures usually met with. A recent case, for instance, had systolic 240 mm., diastolic 160 mm. (J. 12 cm.); with the 5 cm. cuff, systolic 290 to 300 mm., diastolic 195 mm. Excluding nephritis, however, there are fairly numerous cases showing hypertrophied hearts with high arterial pressure, as a

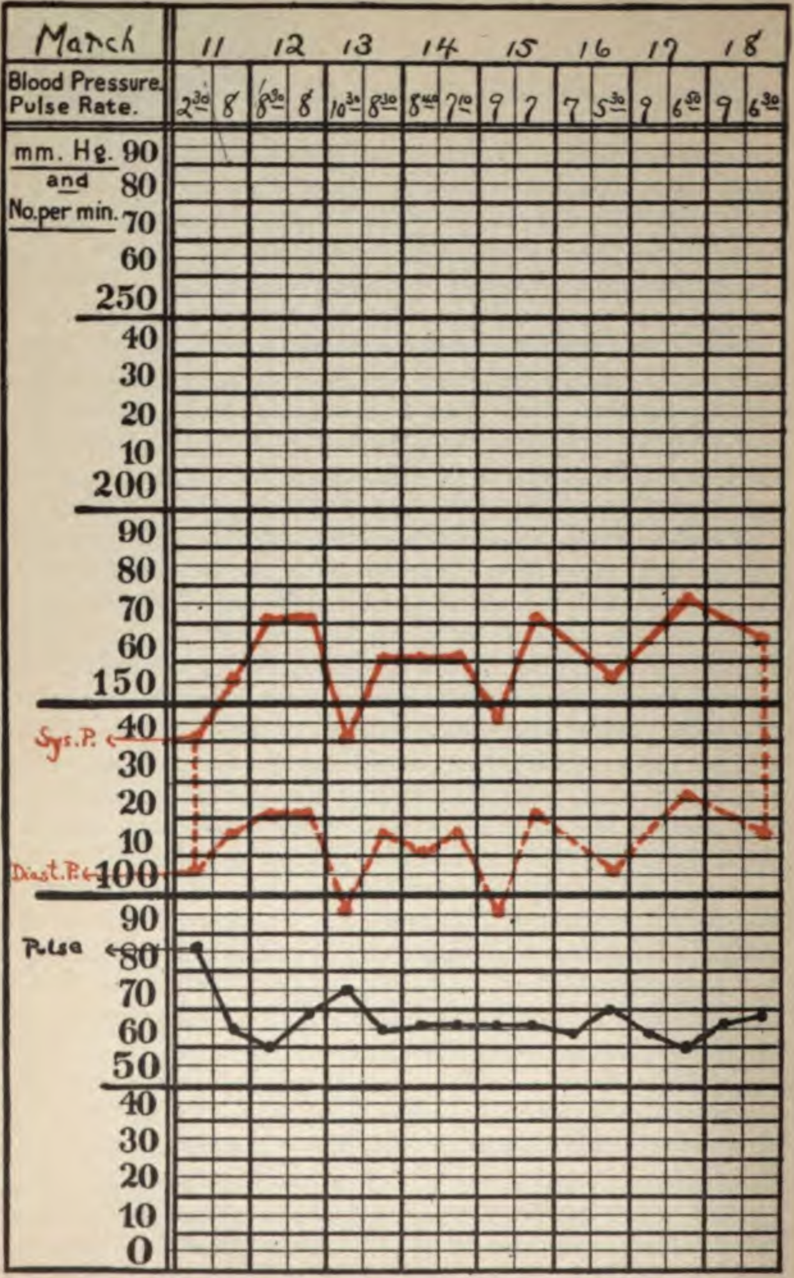

March
11
12
13
14
15
16
17
18
Blood Pressure.
Pulse Rate.
2 30
8
8 30
8
10 30
8 30
8 40
7 10
9
7
7
5 30
9
6 50
9
6 30
mm. Hg. 90
and 80
No. per min. 70
60
250
40
30
20
10
200
90
80
70
60
150
Sys. P.
40
30
20
10
Diast. P.
100
90
Pulse
80
70
60
50
40
30
20
10
0

FIG. 45.

FIG. 45.—GENERAL ARTERIO-SCLEROSIS. (Author's sphygm. 12 cm.)

Chart from male, aged seventy, with marked arterio-sclerosis, general, retinal, and cerebral; cardiac hypertrophy, and slight chronic nephritis; during an attack of acute bronchitis.

Note the moderate hypertension and moderate increase of pulse-pressure.

rule with evident arterial thickening, but sometimes without. These must be considered as having, with or without the changes in the larger arteries, a permanent augmentation of the peripheral resistance in the smaller arteries and arterioles, especially in the splanchnic circulation, or a general narrowing of the larger visceral arteries. This tallies with the pathological findings quoted, and only thus can the absence of high pressure in the ordinary arterio-sclerosis be explained. These cases are the important ones practically, and their recognition is much easier by the sphygmomanometer than in any other way. They are comparatively infrequent in dispensary and hospital experience, where the patients do not apply for treatment until late, and general arterial disease and chronic nephritis usually coincide. Among the well-to-do this form of cardio-vascular disease is frequent and apparently increasing. The name v. Basch proposes for it, angio-sclerosis, seems to me more appropriate than Huchard's designation, presclerosis.

v. Basch's elaborate classification into pseudo-, latent, and manifest angio-sclerosis, and manifest arterio-sclerosis, on the basis largely of the state of the blood-pressure, I think goes beyond the facts. He neglects wholly the possibility of primary muscular disease of the heart, with arterial changes as a later complication; and certainly arterio-sclerosis in all its manifestations cannot be looked upon as a later development of changes in the arterioles. His manifest cases, which show albumin in the urine, must many of them be nephritics. Nevertheless he has done a great service, in calling attention to the fundamental importance of the blood-pressure in the diagnosis and management of diseases of the arteries.

To illustrate the height to which arterio- or angio-sclerosis, with cardiac hypertrophy, may raise blood-pressure independently of any kidney involvement, I will cite two cases, in whom chronic Bright's could be excluded as nearly as may be during life. One gave a reading for systolic pressure of 250 mm., diastolic 180 mm.; the other, systolic 240 mm., diastolic 160 mm. (both Erl. 5 cm.). The highest figures in this condition since using the 12 cm. armlet have been, systolic 205 mm., diastolic 135 mm. This was in a lady, sixty-three years of age. Without some evidence of arterial thickening I have not met a case above systolic 190 mm., diastolic 150 mm. (Erl. 5 cm.). The patient in question was a gentleman of forty-nine, with inherited tendencies to vascular disease, who for a few months had been troubled with palpitation and uncomfortable consciousness of his heart, but who showed simply the moderately high tension, very slight enlargement of the heart, and a sharp aortic second sound, the arteries being imperceptible when empty.

To summarize, we may say that arterio-sclerosis, as a disease of the larger superficial vessels, is without marked influence on blood-pressure; that high blood-pressure argues involvement of the small arteries, especially in the splanchnic circulation, and that such cases should be classed in a separate category; that in all cases systolic pressure is increased much more than diastolic, on account of the lessened distensibility of the arterial walls, due either to the high tension or to the disease, or both.

b. Of the Cerebral Vessels; Apoplexy.—The fact that cerebral arterial disease can in no way be diagnosticated from the general blood-pressure has already been mentioned (see page 143). Patients with the most extreme general arterio-sclerosis may live to old age without cerebral accident, and others, with little disease elsewhere, die of apoplexy in middle life. As a rule, however, cases of hemiplegia, whether due to thrombosis or hæmorrhage, show some peripheral sclerosis and moderate elevation of tension (see Figs. 43 and 62).

Most important is the extreme functional hypertension which large intracerebral hæmorrhage causes. Cook and Briggs, and Cushing have given special prominence to this. The rise thus produced was up to 350 and 400 mm., figures unapproached in any other condition. Just how much error their narrow armlet introduced, it is hard to say. This grade of hypertension, if always present, would serve to differentiate apoplectic coma from any other, except uræmic, and would count somewhat against that. I have not seen such figures; but we do not often meet the acute apoplexies at the City Hospital. Thrombotic softening is the common cause of hemiplegia in our patients, even when they have granular kidneys and high blood-pressure. Cook and Briggs, and Cushing, both lay stress on the danger of hypotensive medication in cerebral hæmorrhage, and believe surgical interference, with evacuation of the clot, the rational treatment. Further discussion will be found in the chapter on blood-pressure in nervous diseases.

c. Other Local Processes—Thrombosis, Aneurism, Compression.—Thrombosis of a subclavian, axillary, or femoral artery is rare, and, when present, is not difficult of detection by the finger. The sphygmomanometer may be helpful in incomplete obstruction, or in following the progress of collateral circulation, by measuring the difference of pressure in the two extremities. In one patient with general arterio-sclerosis and cardiac hypertrophy, without nephritis, the left axillary artery became completely obstructed, with disappearance of the radial pulse, in October. The following June some pulse could be felt, and the sphygmomanometer showed: right arm, systolic pressure 190 mm., diastolic 135 mm.; left arm, systolic 115 mm., diastolic 105 mm. (Erl. 5 cm.). By January the pulse was distinctly fuller and the reading was: right arm, systolic 205 mm., diastolic 135 mm.; left arm, systolic 145 mm., diastolic not obtainable (J. 12 cm.) (see Fig. 46, taken on another day).

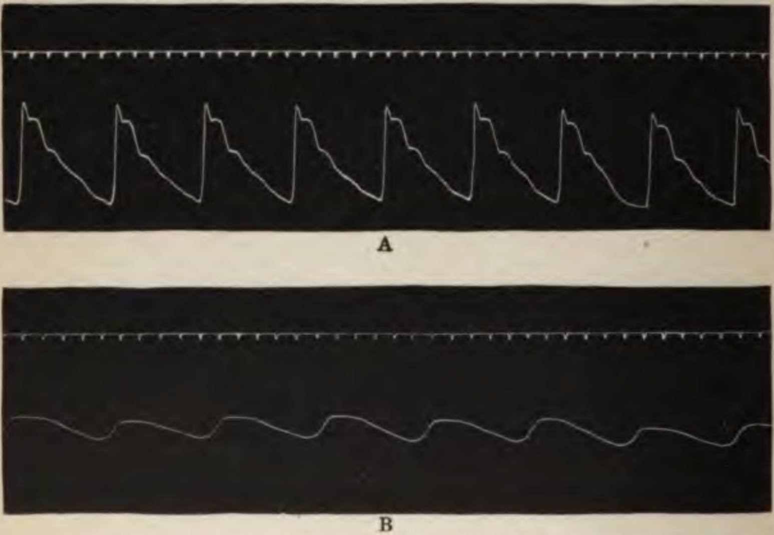


Fig. 46.—Pulsus differens. Case of obstruction of the left axillary artery. (Jaquet's sphygmograph. Author's sphygm. 12 cm.)

A, Tracing from right radial. Systolic pressure, 205 mm., diastolic, 135 mm.
B, Tracing from left radial. Systolic pressure, about 105 mm.

The pulsus differens, occasionally a valuable diagnostic sign of thoracic aneurism, may be detected in its slighter grades in a similar way. It is unwise, however, to admit differences of less than 20 mm. as evidence. Apart from this, the finding of hypertension calls attention to the presence of general or visceral arterial disease, or chronic nephritis, as well as the local condition, since aneurism, of itself, does not affect general blood-pressure. I know of no observations on femoral pressure in the diagnosis of abdominal aneurism, but it might be expected to show a diminution, compared with readings from the brachial. A wide armlet would be essential for this purpose. Similarly, local aneurisms, axillary or brachial, might produce the same effects as the partial occlusion of the artery from other causes.

Ekgren reports an interesting observation on a case of mediastinal tumor. In the standing position systolic blood-pressure was alike in the two arms, but, on lying down, it was invariably 60 to 70 mm. lower in the right arm. Autopsy showed a large mediastinal lympho-sarcoma, involving the right lung, but movable in such manner as to compress the main branches of the aorta on the right side, when the body was on its back.

D. Diseases of the Heart-Muscle.—The clinical diagnosis of muscular heart lesions seems more influenced by prevailing fashions than that of almost any other group of pathological conditions. Not long ago it was the custom to diagnosticate "fatty heart" in all patients showing cardiac enlargement with symptoms of insufficiency, not due to valve defect or kidney disease. Now these same people are usually said to have "chronic myocarditis." I have seldom seen either diagnosis strikingly confirmed at autopsy, though the latter designation comes nearer the truth. Even the pathological diagnosis of a heart-muscle is not easy, and ordinary stained sections are of small value. My personal feeling inclines strongly to the belief that an anatomical diagnosis in these cases will always be beyond the reach of clinical investigation, and worth little when attained. Rolly, Hasenfeld and Fenyvessy, and Pässler and Rolly, have all shown experimentally that considerable myocardial change is compatible with adequate reserve force of the heart. On the other hand, our present methods do not always reveal histological alteration in the muscle commensurate with the functional disturbance, in fatal cases of heart disease.

A fairly accurate functional diagnosis is not only possible, but of real importance for prognosis and treatment. A knowledge of the condition of the blood-pressure is essential to it. With the sphygmomanometer, the cases of large weak heart that seek treatment on account of dyspnœa, precordial distress, cardiac asthma, œdema, or subjective disturbances of rhythm, may be quite sharply divided into two groups; the one with distinct hypertension, the other without. The first group contains the hearts in which hypertrophy is, or is becoming, insufficient to compensate for the increased peripheral resistance. They are, therefore, the cases secondary to kidney disease, arterio- or angio-sclerosis, or are cases of primary myocardial disease which have developed Bright's or arterial changes. In the second are the primary uncomplicated forms of myocardial disease, and sometimes the terminal stage of patients who earlier belonged in the former group. v. Basch has studied the secondary cases exhaustively, and his book contains much that is suggestive. They are by far the more frequent in practice, and the importance of their recognition lies in the prime necessity for treating the condition back of the hypertension.

The following case is typical. A gentleman, fifty years old, of excellent habits (an atypical feature), had noticed slight irregular heart-action ten years previous. Eight years later it recurred, with shortness of breath, while on the ocean. A year later he had dyspnœa and oppression while crossing the Rocky Mountains. At the time of his first visit he had suffered from rapid, irregular heart action, and considerable dyspnœa on exertion and at night. His heart extended one inch beyond the left nipple line, with a large area of absolute dulness. At the apex there was gallop rhythm, and a systolic murmur, which was heard out into axilla and also upward over the heart. The systolic arterial pressure was 260 mm. (R. R. 5 cm.), with a large arm. Urine showed nothing abnormal. He was given advice as to the avoidance of all sudden or forced exertion, moderation in diet, no smoking, etc., and put on potassium iodide, gr. v, after meals, and strychnin, gr. $\frac{1}{50}$, before meals; also nitroglycerin, gr. $\frac{1}{100}$, for emergency use in attacks of dyspnœa. Twelve days later he professed himself feeling perfectly well, but the heart showed no change. Systolic pressure, 230 to 240 mm. Four months later he was having only occasional attacks of precordial distress and dyspnœa at night, relieved at once by nitroglycerin. The heart dulness now came only to the nipple line, but gallop rhythm and murmur persisted. Systolic pressure, 220 to 230 mm. Six months later he was able to walk a mile with perfect comfort, and was about his country place on his feet all day. The heart now showed no gallop rhythm, and only a faint apex murmur when recumbent, none standing. The systolic pressure was 225 mm. He had taken the strychnin and iodide steadily for six months.

Two months after this he was still well, the heart showed no special change, but systolic pressure was 250 mm., diastolic 180 mm. (Erl. 5 cm.). He continued well until fourteen months from the first visit, when he was caught out walking in a severe snow-storm. Following this he had repeated nocturnal attacks of moderate œdema of the lungs, that is, urgent dyspnœa, with rattling in chest, terminating in less than an hour with the expectoration of copious watery secretion. His heart became larger again, rate 100 to 108, systolic pressure 245 mm. The legs showed a little œdema, and the urine contained albumin with hyaline and a few granular casts. There was then evident insufficiency of the right ventricle superadded. He was put on infusion of digitalis, and the iodide and strychnin resumed, with nitroglycerin for emergencies as before. He improved somewhat, but did not regain the comparative comfort he enjoyed before the exertion in the snow-storm overstrained his left heart, and brought on the secondary insufficiency of the right. He died after four months, without my seeing him again.

The patients with primary uncomplicated cardiac insufficiency, whom I have seen, had for the most part high normal pressures, 135 to 145 mm. (12 cm.), 150 to 190 mm. (5 cm.), with fairly normal pulse-pressure. One case showed systolic pressure of 145 mm., with diastolic 120 mm. (J. 12 cm.), but the heart was rapid. These cases without hypertension are more prone to have œdema and evidence of right ventricular failure. Some, however, have anginal pain on exertion. I have once seen very low normal pressure with the former symptoms, which was persistent after marked evidence of a weak right ventricle had disappeared.

The patient was a very active man of fifty-seven, who, four years before, had had a left hemiplegia, from which he made a complete recovery. Two months before he was first seen by my father, he was suddenly seized with such extreme dyspnœa that he thought he should die. He fell on the floor, was found with very feeble pulse, and had hypodermics of nitroglycerin given him. For a month he was in bed with constant orthopnœa, and had double hydrothorax and œdema of the legs. The heart was large, with faint sounds and no murmurs. In spite of his great discomfort he would not keep absolutely quiet; but a month later a mild attack of lobar pneumonia enforced bed, and his improvement began then. On infusion of digitalis and strychnin he made rapid gains, and after a month was able to walk two miles.

At this time his heart was moderately enlarged, but not readily made out, on account of overlapping lung. The left border seemed just beyond the nipple line. The sounds at the apex were faint and in gallop rhythm. At the base the pulmonic second was markedly accentuated. There were no murmurs. Lungs showed a little crackling at base. No fluid in chest, no œdema of legs. Five months later, having had dyspnœa on exertion for a month, since becoming involved in an exciting affair, his heart was the same, and systolic pressure 135 mm. (5 cm.). He was put on digitalis and strychnin again. Now, four months later, he is in fairly good condition; no œdema, no trouble at night, but dyspnœa on exertion. He has taken no care of himself, and is carrying heavy responsibility. His heart now comes scarcely to the nipple line. There are no murmurs. The lungs are clear. The urine contains neither albumin nor casts. Systolic pressure is 105 mm., diastolic 85 mm. (J. 12 cm.); systolic 135 mm. (R. R. 5 cm.) as before. This was evidently a primary insufficiency of the left ventricle preponderantly, with secondary weakness of the right.

Another patient of fifty-one has had an enlarged heart for eleven years, without murmurs, except when it becomes more dilated and develops a temporary mitral murmur. The pulmonic second sound is always extremely accentuated, the action markedly irregular and usually rapid. There is no arterial thickening, and no urinary change, except when his right heart flags. He has had dyspnœa on exertion, and some cyanosis, which have increased of late years. The condition originated in what was probably an acute myocarditis in a rheumatic attack. During the eleven years he has been through several attacks of mild pneumonia, or at least pulmonary congestion with fever; attacks of severe bronchitis; and one serious time of acute cardiac insufficiency, with urgent dyspnœa, œdema, hydrothorax, congested liver, etc., two years ago. He has taken great care of himself as regards exertion and excesses, and uses digitalin, or infusion of digitalis, and strychnin in small doses most of the time. His systolic pressure, for the largest waves, does not go above 145 mm., with the smaller waves about 20 mm. lower; diastolic 105 mm. (J. 12 cm.).

The cases of angina pectoris will be considered by themselves, as well as the measurement of the volume irregularity of the pulse, so common in these patients.

E. Valvular Heart-Disease.—a. Aortic Insufficiency.—Nowhere does the apparent contradiction between results obtained with the instruments measuring systolic, and those measuring diastolic pressure, stand out more clearly than in what one reads about aortic insufficiency. Thus we find Giglioli, using the Riva-Rocci, stating that the pressure is high in compensated cases, as much as 175 mm.; Hayaski, with both Riva-Rocci and Gärtner's tonometer, finding hypertension; and Vaguez, with tonometer, getting readings of 200 to 220 mm. On the other side, Carter found the pressure in uncomplicated aortic insufficiency always low, 80 to 85 mm.; but he worked with the Hill and Barnard sphygmometer. Both are correct observations, and both are entirely erroneous and misleading statements, simply because there has been little knowledge among clinicians of the criteria used for indirect blood-pressure determinations, and all findings alike have been published as observations on "blood-pressure." Potain went even further wrong and tried to explain how, after all, the pulse of aortic insufficiency did not have the characters of low tension. As a matter of fact, the great interest of using the sphygmomanometer in a case of aortic insufficiency is, that it gives a numerical value to the well-known pulsus celer, which expresses perfectly the mechanical effect of the lesion on the systemic arterial circulation. Hensen and the author both called attention to this some years ago, and the advent of clinical instruments which determine both pressures should put a stop to the confusion that has reigned. The actual figures found are striking. One sees in average cases a rise in pressure with each pulse-wave of 75 per cent. of the original diastolic pressure, in more extreme lesions 100 per cent. One patient, for instance, a gentleman of sixty-five, with aortic insufficiency of long standing and practically no aortic second sound, I have measured sixteen times in six weeks, with my sphygmomanometer (12 cm.). His systolic pressure has varied between 130 and 155 mm., diastolic between 60 and 75 mm., and pulse-pressure between 65 and 85 mm. The average has been, systolic 145 mm., diastolic 68 mm., difference 77 mm., an average pulse-pressure 113 per cent. of the previous diastolic pressure (see Fig. 47). This is the largest difference I have ever seen in an uncomplicated case, using a trustworthy method.

Aortic insufficiency with chronic nephritis or arterio-sclerosis may give even greater values for the pulse-pressure. One man with an extreme lesion, of specific origin, with general arterial dilatation and chronic Bright's, who suffered from anginal pain on slight exertion, had a systolic pressure of 220 mm., diastolic 90 mm. (Erl. 5 cm.), difference 110 mm., equal to 122 per cent. of the diastolic pressure. The same patient, three months later, with dilated right heart and dropsy, had a systolic pressure of 270 mm. This shows that, in cases with nephritis at any rate, loss of compensation is not invariably attended by a fall in pressure. More interesting, from a diagnostic standpoint, are the combined valve-lesions, in which the important problem is as to which preponderates. I have not examined many cases of this kind, but all have shown a marked reduction in the pulse-pressure. One patient with a double aortic lesion, in whom the systolic murmur was unusually harsh and pronounced, showed systolic pressure 115 to 120 mm. (slight irregularity), diastolic 80 mm. (J. 12 cm.), pulse-pressure 40 mm., equal to 50 per cent. of the diastolic. A man, on the other hand, with double murmur, but the systolic element softer, had a systolic pressure 200 mm., diastolic 100 mm. (Erl. 5 cm.), pulse-pressure 100 mm., equal to 100 per cent. of the diastolic. This ability to measure the mechanical effect of the leakage on the pulse seems to me of great value in such cases

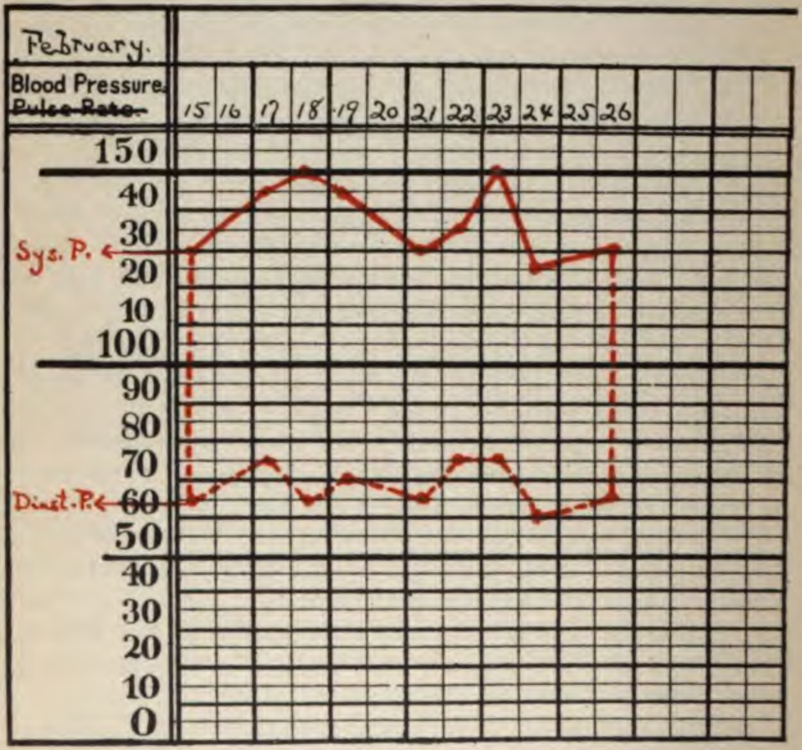


FIG. 47.—AORTIC INSUFFICIENCY. (Author's sphygm. 12 cm.)

Chart from male, aged sixty-five, with extreme aortic insufficiency, fairly compensated. All observations in bed. in the morning.

Note the extreme increase of pulse-pressure and the variability of blood-pressure.

with double aortic murmur, for the extent and direction of cardiac enlargement is the same whether stenosis or insufficiency preponderates. The systolic aortic murmur is usually heard accompanying aortic leakage murmurs, but only in a few cases indicates an actual stenosis. These cases I believe may be picked out by the sphygmomanometer, provided one excludes the very slight defects, and the cases complicated by mitral lesions, or with broken compensation.

Where aortic and mitral murmurs coexist the evidence is equally valuable, not only as showing which is the chief lesion, but also helping to distinguish between a real complicating mitral stenosis and the so-called Flint murmur. The smallest pulse-pressure I have ever seen in aortic regurgitation was in a woman, forty-two years old, who for four years had had a varying amount of dyspnœa, at times cough and bloody expectoration, and a little œdema of the feet, all pointing to mitral disease. At the time, she was about and in good condition. Physical examination revealed a heart enlarged both right and left, with a slight presystolic thrill and short presystolic murmur at the apex, and a blowing aortic diastolic murmur heard all over the precordium. The aortic second sound was preserved, the pulmonic accentuated, especially when lying down. The pulse was small, regular, not distinctly a pulsus celer. The sphygmomanometer showed systolic pressure 115 mm., diastolic 95 mm. (Erl. 5 cm.), pulse-pressure 20 mm., equal to 21 per cent. of the diastolic. Evidently the aortic lesion, in spite of the loud murmur, was insignificant. To contrast with this, a young man with double aortic and mitral systolic murmurs, had systolic 125 mm., diastolic 65 mm. (J. 12 cm.), pulse-pressure 60 mm., equal to 92 per cent. of the diastolic pressure.

The effect of the disturbance of compensation on blood-pressure and pulse-pressure is hard to discuss with positive conviction. I have seen both remain very high where the heart was much dilated, and severe dyspnœa and attacks of cardiac asthma were present. Here the mitral remained sufficient. There is no doubt that systolic pressure may remain high in spite of marked cardiac insufficiency, as Hensen and others have shown. With the development of relative mitral insufficiency, one would expect a reduction in the pulse-pressure, but further studies of systolic and diastolic pressures are needed to clear up this point.

One further feature of the blood-pressure in aortic disease must be recorded, namely, its variability without discoverable cause. This is true especially of the systolic pressure. Hensen records cases where fluctuations of 40 mm. occurred in a few hours. The diastolic pressure maintains a more constant level.

b. Other Valve-Lesions.—The sphygmomanometer is peculiarly disappointing in valvular heart-diseases other than aortic insufficiency. We have already seen how little one may argue from the height of blood-pressure as to the work of the heart (see page 161). There is a general consensus of opinion that the bulk of cardiac cases have pressures within normal limits, whether the measurements be of systolic or diastolic pressure (v. Basch, Potain, Hensen, Hayaski, Norris, Goldwater, Carter, Jarotzny). Studies of the pulse-pressure by reasonably accurate methods are as yet lacking. I have found it as low as 16 per cent. of the diastolic in a patient with aortic stenosis and mitral insufficiency, where the pulsus tardus was very evident. Also in mitral stenosis, especially if one considers the smaller waves rather than the occasional large ones, it may be much diminished. Accurate measurements are scarcely possible by an indirect method, when so much irregularity exists as in these bad mitral cases. One woman, for instance, with double mitral murmur and a much dilated heart, had a diastolic pressure of about 100 mm., with systolic for the small waves 105 mm., for the large 120 mm. Here there was a marked reduction of the pulse-pressure, which varied between 5 per cent. and 20 per cent. of the diastolic. Nevertheless, in another patient with extreme mitral insufficiency of long standing, recovering from a breakdown, and on digitalis, I have repeatedly found systolic pressure 160 to 165 mm., diastolic 110 mm., pulse-pressure 50 mm., equal to 45 per cent. of the diastolic. Further observations along this line are much to be desired.

Concerning the effect of compensation or decompensation in these cases, the blood-pressure seems valueless, at least when systolic or diastolic pressure alone is taken. v. Basch long ago called attention to the fact that systolic pressure may even rise when cardiac insufficiency occurs. Here the dyspnœa, the renal involvement, the mental anxiety, the unnoticed œdema, all come into play. The psychical state, so distressing a feature of heart-disease in its later stages, I think has been insufficiently emphasized as a possible factor in this hypertension. Studies of the effect of morphin, which so often acts beneficially under just such circumstances, might help decide its importance. The fact that hypertension and impending cardiac failure may coexist, only makes it more evident that the sphygmomanometer tells us far more of peripheral resistance than of the heart. Two facts must be borne in mind in reading any statistics concerning valvular lesions. First: Aortic and mitral murmurs in elderly people are frequently due to sclerotic changes in the

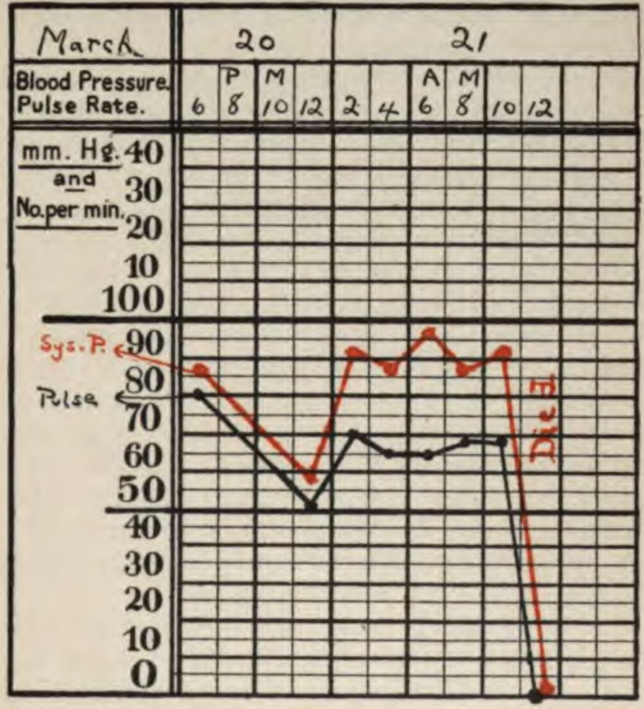


FIG. 48.—TERMINAL HYPOTENSION IN CARDIAC DISEASE.

Chart from T. B., aged thirty-five, male, City Hospital. Old mitral insufficiency and subacute endocarditis with fever. General venous congestion and œdema.

Death from asystole, heart stopping some time before respiration ceased.

Note the parallel movement of pressure and pulse. Contrast with death from collapse (vaso-motor death), as shown in Fig. 59.

valves, which may be accompanied by similar and unnoticed arterial lesions, and hypertension be dependent on the latter. Second: Mitral insufficiency is so commonly due to a dilatation of the mitral ring (muscular or relative insufficiency), that many of the cases reported belong with the muscular, rather than with the valvular diseases. Studies in unquestioned simple valve defects, due to preceding acute rheumatic endocarditis, are needed, before we can conclude that blood-pressure is meaningless in valvular heart-disease.

c. Acute Endocarditis.—In acute endocarditis the only records I can find are those of Shaw. He found subnormal pressures in children with endocarditis complicating acute rheumatism, as low as 80 mm. (G.).

F. Cardiac Neuroses.—a. Exophthalmic Goitre.—Arterial pressure in Graves's disease has generally been considered high by those who used the sphygmomanometer. Hensen, Jackson, and Gross all report cases, and in none of the series was the systolic pressure low, though extreme hypertension (over 200 mm., R. R. 5 cm.) did not occur. My own observations have coincided with this view, but since they were all isolated and not repeated determinations, and patients with this disease are exceedingly nervous, they probably contain a share of psychical hypertension. The recent careful studies of Spiethoff on twenty cases with the Riva-Rocci apparatus, and v. Recklinghausen armlet for comparison, have led him to the following conclusions:

"1. That the blood-pressure shows no constant change in either direction in Basedow's disease; the views that the pressure is always raised, or that it is always lowered, are equally inadequate."

"2. Lowering of pressure and high pressure are both found in the severe cases, while the lighter forms do not vary much from the normal."

He cannot say definitely whether the heart or the vasomotor system is responsible for the variation. Neither pressure nor pulse-rate were satisfactory indications of the severity of the disease.

Gross calls attention to the great lability of the blood-pressure in these patients, either from slight mental causes, or apparently spontaneously. This is easily comprehended, when such overirritability of the whole nervous system exists. Clinical observers have been in the habit of considering tension diminished in this disease. I believe the discrepancy between this and the usual objective determinations finds its explanation in the same cause as aortic insufficiency—the marked elevation of systolic pressure and pulse-pressure, diastolic pressure not rising equally. The sphygmographic trace obtained in Graves's disease is often an outspoken pulsus celer. Unfortunately the rapidity of the pulse and the muscular tremor make estimations of diastolic pressure very difficult in these people, and I can only record a single satisfactory one. Here the systolic pressure was 200 mm., diastolic 130 mm. (Erl. 5 cm.), pulse-pressure 70 mm., equal to 54 per cent. of the diastolic. Were all the pressures corrected for the narrow armlet, this would be about 65 per cent. In this case, slight perturbation ran up the systolic pressure to 240 mm.

b. Other Nervous Disturbances.—The classification of these obscure conditions is unsatisfactory. Anything, from simple palpitation in young girls to fatal angina pectoris due to coronary thrombosis, may be forced into the category. The only work relating especially to these so-called neuroses is that of Hochhaus. He claims to have found high pressures, in twenty men 150 to 210 mm., in sixteen women 140 to 210 mm. (narrow armlet of course), all with neuroses. He propounds the thesis that hypertension is diagnostic of nervous, as against organic affections of the heart. His neuroses were either sensory, with precordial oppression, or even severe anginal pain, as its evidence; or motor, with tachycardia or arrhythmia.

I am absolutely unwilling to accept his conclusions. His sensory neuroses, I believe, all belong with the clinical group of myocardial diseases, in which hypertension is the evidence of permanent changes in the small vessels. Many of the cases with disturbance of rate or rhythm also belong in the same category, if the tension was high, or else were excited when the determination was made. I have never seen a case of paroxysmal tachycardia, a pure neurosis, if there be such, with anything but normal or low normal pressure between the attacks. During attacks I have made few observations, but they have not shown hypertension. As to the arrhythmias, they are most frequent in elderly people, where one always suspects latent angio-sclerosis. Those I have examined, in which no proof of organic disease could be obtained, had pressures below 160 mm. (R. R. 5 cm.). Hypertension with subjective cardiac disturbance is, I believe, distinct warning of the onset of vascular disease.

G. Important Cardio-vascular Symptoms.—It is impossible to consider, within a reasonable space, the relation of all the clinical features of cardio-vascular disease to the blood-pressure. Practically all of them are due rather to the condition of the neuro-muscular apparatus of the heart relative to the demands upon it, than to the absolute amount of those demands. The chief importance of a knowledge of the blood-pressure in connection with dyspnœa, or distress on exertion, or other evidence of heart disturbance, lies in its testimony as to the existence of an unnecessarily high peripheral resistance, which may be combatted. Angina pectoris, however, is a symptom-complex so important and so distinct, that it deserves separate consideration. Some definite information also exists as to the relation of certain forms of dyspnœa to blood-pressure. Finally, the volume irregularities of the pulse are well studied by the sphygmomanometer.

a. Angina Pectoris.—A certain feeling of distress or pressure beneath the sternum is very common in patients with marked essential hypertension, irrespective of the primary disease. Few of them with pressure permanently above 200 mm. (12 cm.), 220 to 240 mm. (5 cm.), can even walk briskly without it. This is most apt to come with exertion following a meal, and, as belching of gas is a frequent accompaniment, both patient and physician commonly put the blame on the stomach. In more developed form, exercise brings a feeling of intense pressure across the upper chest, as though it were held in a vice, or there is a pain which centres there and radiates to the left or both shoulders, down one or both arms to elbow or wrist, and up the neck to the angle of the jaw. With this the patient feels compelled to stand still, and, unless the attack be of great severity, the sensations pass off in a few minutes and he can walk again. The worst form may come spontaneously, especially at night, as well as after effort and excitement, is of longer duration, and associated with pallor of the face and great mental agony. These are the seizures which often prove fatal. Many deny the name of angina pectoris to the lighter attacks. Nevertheless, the gradual development of these lesser feelings of tightness or distress into the worst pain, which one may frequently follow during the course of years, leaves little room for a nomenclature which attempts to draw a sharp line between them. Perhaps the designation "anginoid" describes the minor attacks as well as any.

In the diagnosis of true angina, or anginoid attacks, well-marked hypertension is important evidence. Though some cardiac enlargement and a systolic aortic murmur, less frequently a mitral murmur, are common findings, and, in connection with the patient's account of his sensations, stamp the attacks as really of this nature; yet, in a certain number, no anatomical change is discoverable by ordinary physical diagnostic methods. If, then, a blood-pressure of over 180 mm. (12 cm. or G.), 200 mm. (5 cm.), be found, anginoid pain can be held due to definite organic disease. The negative evidence is not so convincing. Especially is this true of the severe attacks. I have seen a number of cases of true angina pectoris without high pressure, that is, less than 170 mm. (R. R. 5 cm.). Of these at least three have proved fatal. In one, who died during an attack, post-mortem by Dr. Coplin, of Philadelphia, showed chronic myocarditis with enormous increase in elastic tissue throughout the heart. There were no cardiac symptoms except the pain on exertion, substernal and radiating to the left shoulder and neck, which began six years before. No physical signs of disease were present, either in the way of enlarged area or murmurs. The lowest systolic pressure I have recorded in this condition was 120 mm. (R. R. 5 cm.). The others have varied from 140 to 170 mm. v. Basch notes this finding of low pressure in patients with anginal symptoms, which he has seen in eighteen of his series, forty-seven having had tension above the normal. During the attack the pressure might either rise or fall. In such patients the diagnosis and prognosis depend entirely upon the character of the attacks, and the amount of exertion required to initiate them. With intense pain regularly following slight exertion, and pallor of the face during the attack, a low blood-pressure seems to argue a worse outlook.

Where the description of the pain is atypical, and there seems a reasonable question of its being a neuralgia or a reflex gastric pain, especially when exercise may be taken without difficulty, normal blood-pressure is corroborative testimony that the symptoms are not associated with cardio-vascular disease, and the prognosis may be good. Great care must be used in deciding on these cases, nevertheless.

The case recorded on page 176 illustrates the ordinary anginoid type with hypertension. The following history is instructive as showing the sudden development of intense angina pectoris after repeated slighter attacks. A gentleman, sixty-one, who had been a very hard worker, liberal in his diet and use of stimulants and tobacco, had noticed for a year that fast walking gave him distress across the upper sternum, and slight dyspnœa. As the distress would pass off when he slacked his speed, he paid no attention to it, beyond reducing his ordinary gait. After some months the attacks became more frequent and severe, and followed slighter exertion. In March he consulted Dr. Osler, who gave him amyl nitrite for emergency use, and cautioned him to avoid all strain. Shortly after this, while on a railway train, he was seized with excruciating pain in the chest, running down both arms to the elbow. He broke off his journey, and for three weeks was unable to lie down without bringing on intense pain. He had slight œdema of the legs at that time. With the continued use of nitroglycerin, $\frac{18}{100}$ gr. a day, he improved, and four months later could walk half a mile on the level. He said that, when the pain would come on, he felt as though he would stop breathing and drop. Physical examination, four months after the intense seizure, showed merely faint heart sounds with a very slight basic systolic murmur. As he weighed two hundred and forty pounds, no satisfactory percussion was possible. Heart action was slow and regular. Systolic pressure was only 165 mm. (R. R. 5 cm.), distinctly low for the size of arm. The urine showed a few hyaline casts only. Two years later he was living and attending to business.

Another patient shows the possibility of a relatively good prognosis. He is sixty years old, and for nine years has suffered some precordial distress on exertion. For four years there has been distinct anginal pain at times, always after effort. Two years ago he was obliged to climb a short but steep incline in winter, and had intense angina pectoris; but subsequently he has never had a severe attack. Now, walking against the wind, or a fit of anger, precipitates an anginoid seizure, but nothing like his past ones. He has a simple hypertrophy of the heart, without murmurs or nephritis, a typical angio-sclerosis, or cardio-sclerosis, as Huchard would put it. His systolic pressure is 215 mm., diastolic 145 mm. (5 cm.). He improved very much on continued small doses of iodide, and nitroglycerin relieves his attacks promptly.

b. Cardiac Asthma and Cheyne-Stokes Breathing.—The spontaneous attacks of dyspnœa so frequent in cardiac and nephritic cases are very commonly associated with hypertension. In their most severe form actual œdema of the lungs occurs, and the danger to life is extreme, as in the cases previously mentioned. Fig. 49 shows an observation I was able to make during an intense seizure. Since Cohnheim and Welch (see page 150) showed that a paralysis of the left ventricle, with the right still beating, was the mechanical cause of pulmonary œdema, where hypertension exists it demands immediate relief, to lessen the work of the weakened left heart, before cardiac stimulants can be safely used. The sphygmomanometer in this condition gives important therapeutic indications. As the danger is urgent, the rapidly acting amyl nitrite or nitroglycerin are the best drugs, though even these may be disappointing. The severer forms of dyspnœa may also be a cause of hypertension, as v. Basch and Hensen have shown. The effect is of course through the vaso-constrictor centres, as we have seen in experimental asphyxia (see page 37).

In Cheyne-Stokes breathing, variation in the size of the pulse with the periodic changes in respiration is usually observed. Gumprecht noted it, and Norris found the pressure as much as 25 mm. lower in the period of apnœa. The phenomenon is probably caused by variation in the tonus of the vaso-motor centre, synchronous with that of the respiratory centre, due to partial anæmia of the medulla (see page 140).

c. **The Arrhythmias.**—Irregularities of the pulse affect both time and volume rhythm. Only the latter lend themselves to sphygmomanometric study. When present they make observations difficult, especially for diastolic pressure. I am accustomed to note separately the systolic pressure of the largest and of the smallest pulse-waves, and the approximate diastolic pressure. In this way a permanent record of the absolute amount of volume irregularity is obtained. I have seen as much as 40 to 60 mm. difference in systolic pressure within a few seconds, in cases of mitral stenosis or myocardial disease. The smaller variations of 10 to 20 mm. are usually overlooked without the sphygmomanometer. This brings them into relief, because, while maintaining the pressure at just sufficient to obliterate the average pulses, occasional fuller ones will come through in a striking way. In true bradycardia, or the pulsus alternans, the difference between systolic and diastolic pressure

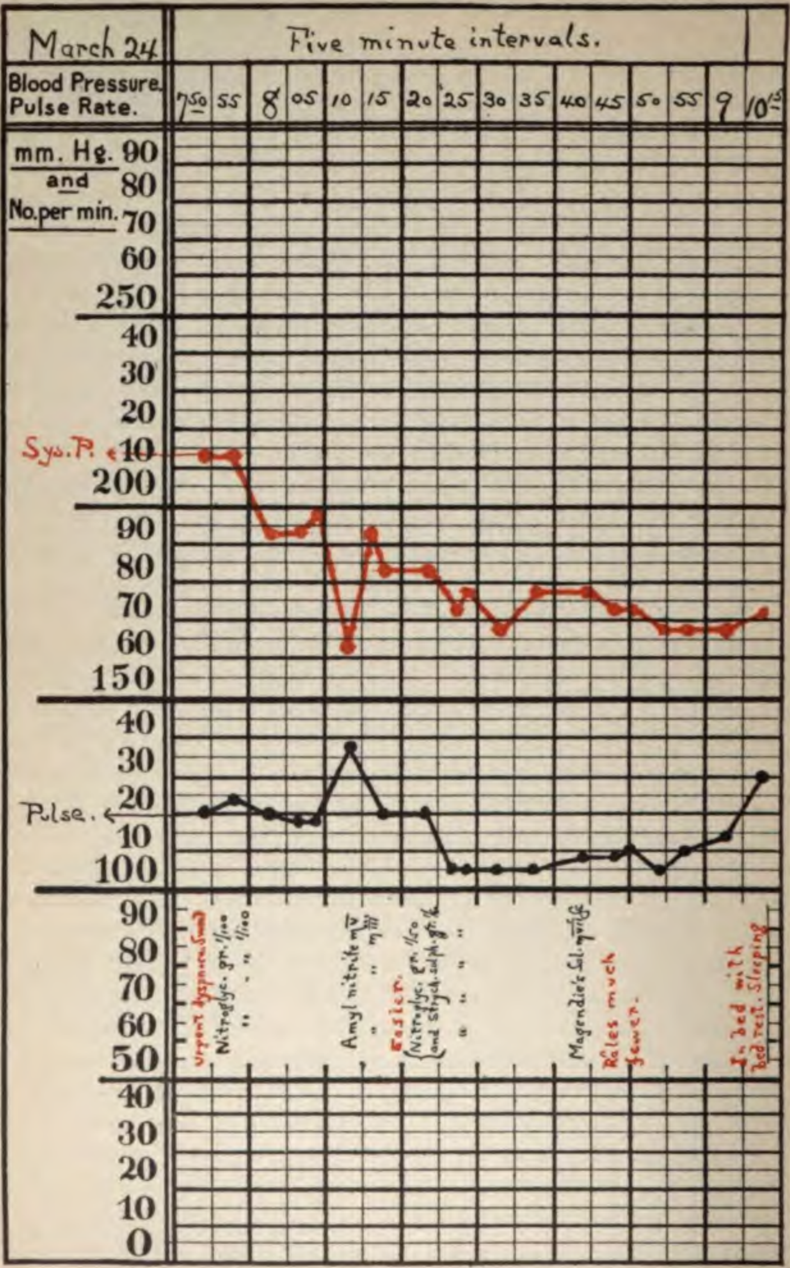

March 24
Five minute intervals.
Blood Pressure.
Pulse Rate.
7^50 55 8 05 10 15 20 25 30 35 40 45 50 55 9 10^15
mm. Hg. and No. per min.
90 80 70 60 250 40 30 20 10 200 90 80 70 60 150 40 30 20 10 100 90 80 70 60 50 40 30 20 10 0
Sys. P.
Pulse.
Urgent dyspnoea. Sweat
Nitroglyc. gr. 1/100
" " 1/100
Amyl nitrite m V
" " m iii
Easier.
Nitroglyc. gr. 1/50 and strych. sulph. gr.
" " "
Magendie's Sol. m viii
Râles much fewer.
In bed with bed-rest. Sleeping

FIG. 49.

FIG. 49.—ŒDEMA OF THE LUNGS. (Author's sphygm. 12 cm.)

Chart from W. O'B., aged twenty-eight, male, City Hospital. Admitted March 19th, with slight gouty attack and chronic nephritis. Pressure on admission 120 mm.

Acute general œdema of the lungs began just before 7.50 P. M., and ushered in a rapidly fatal pneumonia.

Note the slight and transient effect of the vaso-dilators.

is greatly increased, as I have personally observed. In this condition, as in aortic insufficiency, the measurement of systolic pressure alone shows a false hypertension.

The paradoxical pulse, and slighter grades of inspiratory fall, in systolic pressure, have been especially studied by Hensen. He considers them alike signs of circulatory weakness. The lesser grades can only be detected with the sphygmomanometer, and are found in uncompensated heart lesions, pleural effusion, and even at times during febrile disease. As terminal phenomena they are common. The marked paradoxical pulse of Riegel, in laryngeal stenosis, pericardial adhesions or effusions, etc., is palpable as well as measurable. Hensen saw a difference of 40 mm. between inspiratory and expiratory pressure in laryngeal diphtheria. In my experience it has been most frequent with asthma or marked emphysema. Complete absence of pulse during inspiration I saw recently in a man with pericardial and left pleural effusion.

H. Effect of Exertion on Blood-Pressure in Cardio-vascular Diseases.—Our estimate of the functional ability of a heart is usually more or less roughly made from the symptoms developed under the exertion of daily life. The study of the blood-pressure and pulse-rate, during the performance of a measured amount of work, ought to render our judgments more accurate. This has been done, at the suggestion of Dehio, by Moritz for cardiac patients, in the same way as by Masing for the aged. Dehio's apparatus is shown in Fig. 50, and can be easily arranged in any hospital. The work is performed by one leg, in time with a metronome, and pressure and pulse measurements from the arm are not interfered with. In Dehio's investigations a weight of 4,090 grams, about eight pounds, was lifted 40 cm. = 15¾ inches, fifty times a minute, giving an actual performance of 81.8 kilogrammetres a minute. A weight of five pounds lifted a foot would seem to me a good average, and I am arranging such an apparatus at the City Hospital. The work from this will be five foot-pounds at each stroke. Pressure determinations should be at three- to five-minute intervals. Moritz's results, in cases of cardiac weakness, are similar to Masing's for the aged (whom Dehio believes have "myofibrosis cordis"), and Fig. 40 illustrates the typical curve, save for the slow senile pulse. The initial rise in pressure was more pronounced than in the normal man, but was not maintained, a fall occurring during the exertion. In the normal persons a steady level of pressure continues throughout, unless the work be very excessive. With the falling pressure, dyspnœa and distress developed. Butterman made similar observations with the tonometer and work in a Gärtner ergostat. Compensated mitral lesions, nervous hearts, etc., reacted with a normal rise, averaging 13 mm. They had no great dyspnœa. Other patients with myocardial disease, who had much distress on exertion, showed a diminution in tension of 3 to 14 mm. Schott found a marked fall in pressure when lasting dyspnœa appeared. This simple method of functional heart diagnosis deserves a careful trial by those who have to advise cardiac patients as to the ordering of their daily life. Exercise, if not

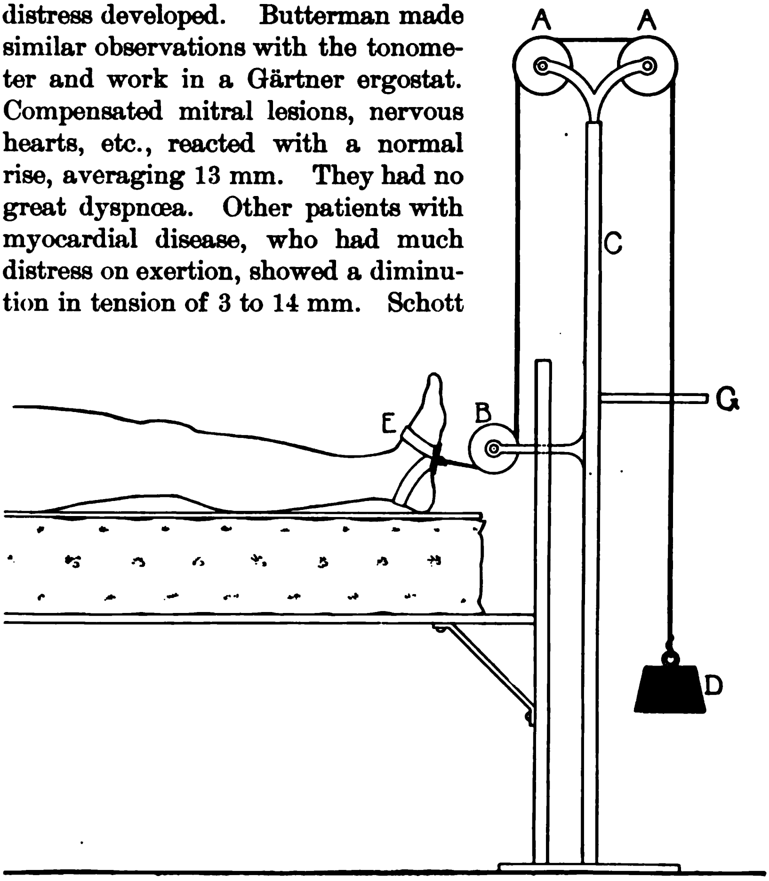


FIG. 50.—DEHIO'S APPARATUS. (From Dehio.)

For performing a measured amount of work with the legs.

The cord, which holds the weight (D), passes over the pulleys (A A), and under the pulley (B), fastened to the upright (C), and hooks into the foot-bandage (E). The height of the stop (G) fixes the distance the weight travels.

harmful to their hearts, is so advantageous for their general health and happiness, that a more accurate method of judging its effects seems a practical desideratum.

I. Therapeutics.—The treatment of diseases of the cardiovascular system has, as its primary object, the improvement of the capillary circulation, so that it may be adequate to the demands of the tissues for oxygen and nutriment, and for the removal of waste products. The ability of the heart to maintain an adequate circulation may be complete during repose, but most deficient during forced exertion. The regulation of the patient's entire life becomes, then, the starting-point of treatment, and should include the avoidance of all those forms of exertion and excitement which cause considerable increase of arterial pressure, especially of a sudden nature. Of the possibility of following the results of this general hygienic treatment with the sphygmomanometer I am unwilling to speak with conviction. The effects would only be evident after a considerable lapse of time, and other causes for blood-pressure variation would complicate the situation. The attempt, however, should certainly be made.

Of the various dietetic and physical measures, massage, baths, etc., I do not feel competent to speak. The good which they do must also be a rather permanent one, of a nature hard to follow by the blood-pressure. Schott has written on the effect of the Nauheim baths on blood-pressure, but to correlate his findings and the benefit received by the patient is another matter. v. Basch and Huchard lay great stress on these methods of combatting hypertension, and I think rightly; but it must be more with a view to limiting a process already initiated, than of causing its disappearance.

As we have already seen, in discussing the value of the blood-pressure as a guide to the functional ability of the heart and circulation, it gives us scarcely any clue to the work performed by the heart. Hence we find, in the records of blood-pressure during treatment of uncompensated heart-lesions, every variation. Christeller, Frenkel, Heike, Hensen, Gross, and Potain, all fail to find any relation between the arterial tension and the circulatory improvement from digitalis. This point is important, for it makes it clear that the effect of many circulatory drugs, of undoubted stimulant properties, will not be visible on the pressure chart; because altered blood-distribution, and not increased blood-pressure, is their best result. In hypotensive states, their effect on pressure is more evident. The main value of sphygmomanometric observations, during the treatment of cardiac cases, is in the avoidance of undue hypertension.

The effect of the vaso-dilators, on the other hand, may and should be studied. Nitroglycerin, amyl nitrite, sodium nitrite, and even spiritus etheris nitrosi, produce an evident fall in pressure. The first two act with surprising rapidity and vigor, but their effect is very transient (see Figs. 42, 44, 49, 51 and 52). Carter found marked and more permanent effects from sodium nitrite, the pressure remaining low for an hour or so. Without blood-pressure measurements, clinicians forget the evanescent action of most of these drugs. Iodide of potassium,

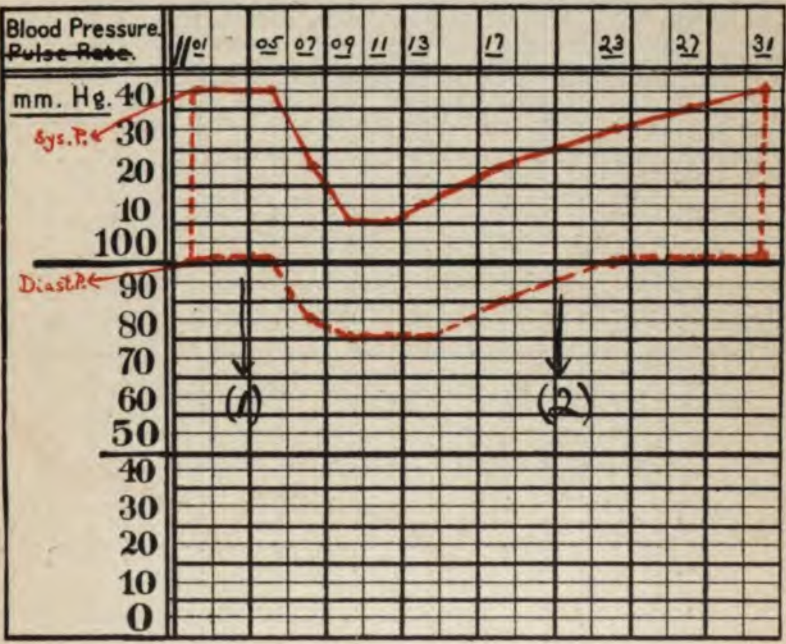


FIG. 51.—FALL IN BLOOD-PRESSURE PRODUCED BY NITROGLYCERIN, gr. 1/100. (Author's sphygm. 12 cm.)

Chart from same case of general arterio-sclerosis as Fig. 45.
(1) Nitroglycerin, gr. 1/100, dissolved in mouth.
(2) Caffein-sodium salicylate, gr. ij, by mouth.

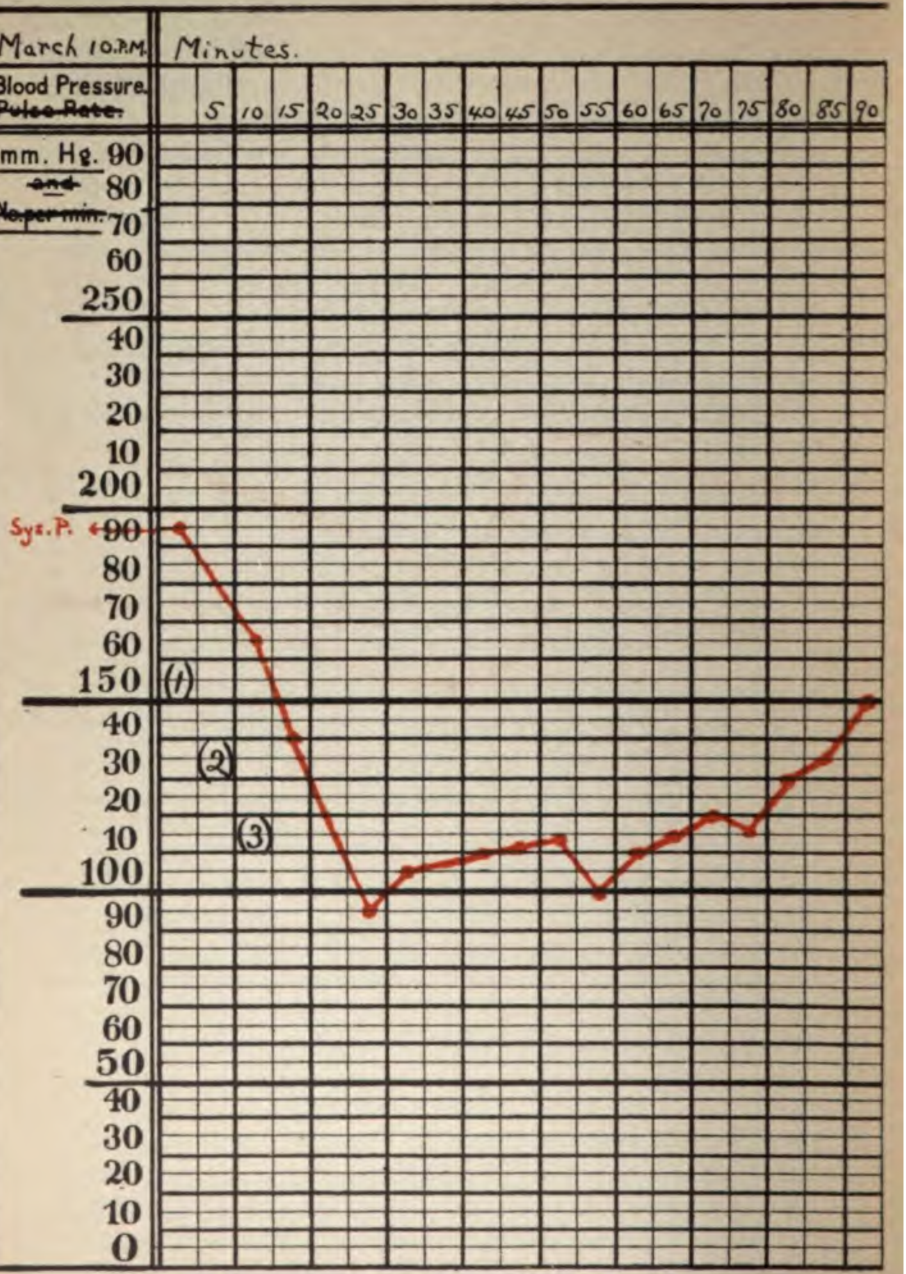

March 10.P.M.
Minutes.
Blood Pressure.
~~Pulse Rate.~~
5
10
15
20
25
30
35
40
45
50
55
60
65
70
75
80
85
90
mm. Hg.
~~and~~
~~No. per min.~~
90
80
70
60
250
40
30
20
10
200
Syz. P.
90
80
70
60
150
(1)
40
30
(2)
20
10
(3)
100
90
80
70
60
50
40
30
20
10
0

Fig. 52.

FIG. 52.—FALL IN BLOOD-PRESSURE PRODUCED BY NITROGLYCERIN, gr. $^3/_{50}$.
(Author's sphygm. 12 cm.)

Chart from same case of chronic interstitial nephritis as Fig. 42.

(1), (2), and (3). Nitroglycerin, gr. $^1/_{50}$, given by hypodermic, for relief of severe nose-bleed. When the pressure reached 95 mm. patient was light-headed and sweating, and nose-bleed had ceased. Immediately after, she went to sleep.

(Observation by Dr. Higgins.)

as a vaso-dilator, is the subject of great dispute. It certainly has not the immediate measurable effect of the nitrites, and cannot be classed as an active vaso-dilator. Nevertheless, I have seen relief of anginal pain on exertion so frequently follow its continued use in small doses, with an apparently lower level of tension, that I am convinced that it has an effect comparable with the hygienic and dietetic measures, when long periods are considered. Some of the cases cited earlier show this. Huchard vaunts its virtues in cardio-sclerosis; v. Basch thinks it of small value. One must always remember, also, that essential hypertension may be a compensatory phenomenon, and, if the anatomical or functional cause of increased peripheral resistance is permanent, it may be most unwise to attempt to lower blood-pressure. In kidney diseases, for instance, lowered pressure may be the cause of diminished blood-flow through the kidney and decreased urinary excretion, an undesirable result.

The most important therapeutic indications of the sphygmomanometer are in conditions of dangerous hypertension, such as uræmia, and in paroxysms of angina pectoris. Here rapid vaso-dilators are called for. In uræmia, the other methods for reducing tension come into play. Venesection is one of the best, and Butterman, Carter, and Hayaski, all report marked reduction in pressure from it. Sweating has a similar effect, and is commonly attended by a fall in pressure, so that the double purpose of reducing pressure and eliminating poisons is achieved. Purgation may also assist. In the light of recent investigation, the depleting treatment of hypertension dependent on acute cerebral compression must remain questionable. Its effects should certainly be controlled by blood-pressure determinations.

LITERATURE

Ascoli, G. Vorlesungen über Urämie. Jena, 1902, pp. 113–117.

v. Basch, S. Die Herzkrankheiten bei Arteriosclerose. Berlin, 1901.

—— Der Sphygmomanometer und seine Verwerthung in der Praxis. Berl. klin. Wochenschr., 1887, vol. xxiv, p. 179.

Butterman. Einige Beobachtungen über das Verhalten des Blutdrucks bei Kranken. Deutsch. Arch. f. klin. Med., 1902, vol. lxxiv, p. 1.

Carter, H. W. Clinical Observations on Blood-pressure. Am. Jour. of the Med. Sci., 1901, vol. cxxii, p. 854.

Christeller. Ueber Blutdruckmessungen an Menschen unter pathologischen Verhältnissen. Zeitschr. f. klin. Med., 1881, vol. iii, p. 33.

Cook, H. W., and Briggs, J. B. Clinical Observations on Blood-pressure. Johns Hopkins Hosp. Rep., 1903, vol. xi, p. 451.

Cushing, H. W. The Blood-pressure Reaction of Acute Cerebral Compression, Illustrated by Cases of Intracerebral Hæmorrhage. Am. Jour. of the Med. Sci., 1903, vol. cxxv, p. 1017.

Dehio, K. Ueber das Altern des Herzens. St. Petersburg med. Wochenschr., 1901, vol. xviii, p. 79.

Edel, P. Ueber die Abhängigkeit der "cyklischen" Albuminurie von der Cirkulation. Deutsch. med. Wochenschr., 1902, vol. xxix, pp. 639 and 663.

Ekgren, E. Ein eigenartiger Blutdruckbefund bei einem Falle von Mediastinaltumor. Fortsch. d. Med., 1902, vol. xx, p. 4.

Erlanger, J., and Hooker, D. R. The Relation of Blood-pressure and Pulse-pressure to the Secretion of Urine and to the Secretion of Albumin in a case of So-called Physiological Albuminuria. Am. Jour. of Physiol., 1904, vol. x, Proceed. of Am. Physiol. Soc., p. xvi.

Forlanini. Rendiconti del R. 1st. Lombardo, 1901. Quoted by Ascoli.

Frenkel, S. Klinische Untersuchungen über die Wirkung von Coffein, u. s. w. Deutsch. Arch. f. klin. Med., 1890, vol. xlvi, p. 542.

Giglioli, G. T. Alcune critiche e alcune richerche di sfigmomanometria clinica. Riv. crit. di clin. Med., 1900, vol. i, pp. 625 and 641.

Goldwater, S. S. Notes on Blood-pressure in Man. Med. News, New York, 1903, vol. lxxxii, p. 926.

Gross, A. Zur Kenntniss der pathologischen Blutdruckänderungen nach Beobachtungen von weil. Dr. H. Hensen. Deutsch. Arch. f. klin. Med., 1902, vol. lxxiv, p. 296.

Gumprecht. Experimentelle und klinische Prüfung des Riva-Rocci'schen Sphygmomanometers. Zeitschr. f. klin. Med., 1900, vol. xxxix, p. 377.

Hayaski, T. Vergleichende Blutdruckmessungen an Gesunden und Kranken mit den Apparaten von Gärtner, Riva-Rocci und Frey. Inaug. Dissert., Erlangen, 1901.

Heike, Wilhelm. Blutdruckmessungen nach Verabreichung von Digitalis, ausgeführt mittels des Riva-Rocci'schen Sphygmomanometers. Inaug. Dissert., Halle, 1901, Aug.

Hensen, H. Beiträge zur Physiologie und Pathologie des Blutdrucks. Deutsch. Arch. f. klin. Med., 1900, vol. lxvii, p. 438.

Hochhaus, H. Ueber functionelle Herzkrankheiten. Deutsch. med. Wochenschr., 1900, vol. xxvi, p. 701.

Huchard, H. La médication hypotensive. Paris, 1903.

Jackson, J. M. A Few Remarks on Blood-pressure. Boston Med. and Surg. Jour., 1903, vol. cxlviii, p. 223.

Jarotzny, A. Zur Methodik der klinischen Blutdruckmessung. Centralbl. f. in. Med., 1901, vol. xxii, p. 599.

Jourdin, Ch., et Fischer, G. De l'importance pronostique et thérapeutique de la pression artérielle Rev. de Med., 1902, vol. xxii, p. 945.

Kaufmann, J., und de Bary, W. Ueber die Einwirkungen Priessnitz'scher Einwickelungen auf den Blutdruck bei croupöser Pneumonie und diffuser Nephritis. Berl. klin. Wochenschr., 1888, vol. xxv, p. 557.

Laqueur, A. Zur Kenntniss urämischer Zustände. Deutsch. med. Wochenschr., 1901, vol. xxvii, p. 744.

Masing, Ernst. Ueber das Verhalten des Blutdrucks des jungen und des bejahrten Menschen bei Muskelarbeit. Deutsch. Arch. f. klin. Med., 1902, vol. lxxiv, p. 255.

Moritz, O. Der Blutdruck bei Körperarbeit gesunder und herzkranker Individuen. Deutsch. Arch. f. klin. Med., 1903, vol. lxxvii, p. 339.

Norris, G. W. A Contribution to the Study of the Human Blood-pressure in some Pathological Conditions. Am. Jour. of the Med. Sci., 1903, vol. cxxv, p. 888.

Pal, J. Zur Pathogenese der akuten transitorischen Amaurose bei Bleikolik, Urämie und Eklampsie. Centralbl. f. in. Med., 1903, vol. xxiv, p. 417.

—— Ueber Gefässkrisen und deren Beziehungen zu den Magen und Bauchkrisen bei Tabiker. Münch. med. Wochenschr., 1903, vol. L^2, p. 2135.

Potain, C. La pression arterielle de l'homme a l'état normal et pathologique. Paris, 1902.

Schott, Theodore. On Blood-pressure under the Influence of Acute Overstraining of the Heart. New York Med. Jour., 1902, vol. lxxv, p. 705.

—— Tonometric Examinations in Chronic Diseases of the Heart. Med. Record, New York, 1901, vol. lix, p. 1028.

Schüle. Ueber Blutdruckmessungen mit dem Tonometer von Gärtner. Berl. klin. Wochenschr., 1900, vol. xxxvii, p. 726.

Shaw, H. L. K. Arterial Tension in Childhood. Albany Med. Ann., 1900, vol. xxi, p. 88.

Spiethoff, B. Blutdruckmessungen bei Morbus Basedow. Centralbl. f. in. Med., 1902, vol. xxiii, p. 849.

Vaguez, M. H. Sphygmomanométrie clinique. Bull. méd., 1903, vol. xvii^1, p. 279.

2. ACUTE INFECTIOUS DISEASES

In acute infections, the main ground for the advocacy of routine blood-pressure estimations must be sought in the experimental evidence of the vaso-motor origin of collapse. Any means which will help to foretell its dread appearance, if in but a fraction of the cases we meet, is well worth while. The sphygmomanometer would seem to offer this prospect, but its absolute value has not yet been determined. For diagnosis it naturally means little or nothing, but for prognosis, and as a guide to treatment, there is good ground for believing that it will come more and more to the front.

Single observations are meaningless in these conditions; only careful records, as of the temperature and pulse, can give the desired information. Any such grouping of diseases on the basis of their typical blood-pressure, as Potain carries it out, is worthless for the acute diseases, though we may distinguish a difference in the regularity with which they affect pressure. The greatest hindrance to determining the real use of the sphygmomanometer in febrile conditions to-day is the lack of sufficiently trustworthy facts. The bulk of the studies have been made with the Potain or v. Basch instruments, many even with the impossible one of Verdin.

A. Typhoid Fever.—In typhoid fever, more than in any other acute disease, the sphygmomanometer has proved its worth. As a guide to treatment, as a help in the diagnosis of perforation and its differentiation from concealed hæmorrhage, and because it affords an additional prognostic standpoint, it should be generally used. We owe especial gratitude to Crile, and to Cook and Briggs, for our knowledge, though some of their results may fail of universal confirmation. To be of any service, blood-pressure must be recorded at the same intervals as temperature and pulse-rate, best on charts. Those who, like Norris, find no prognostic value, made observations once in a few days only. Used in such a way the thermometer would be equally valueless. Typhoid fever is almost invariably hypotensive, as Potain in 116 cases, Crile in 115, and Alezais and Francois in 150, proved conclusively. Unfortunately the latter observers used a very defective method (Verdin's sphygmometer, see page 45), and Potain's own is not free from suspicion. The fall in pressure begins in the first or second week and goes hand in hand with the development of the toxæmia. The absolute pressure means little of itself. The important matter is its general course. Gumprecht calls attention to the marked variations which occur, as they do in a normal man, throughout the day. Alezais and Francois's statements on this point cannot be accepted on account of the instrument used. I think, however, that fluctuations of short duration, up to

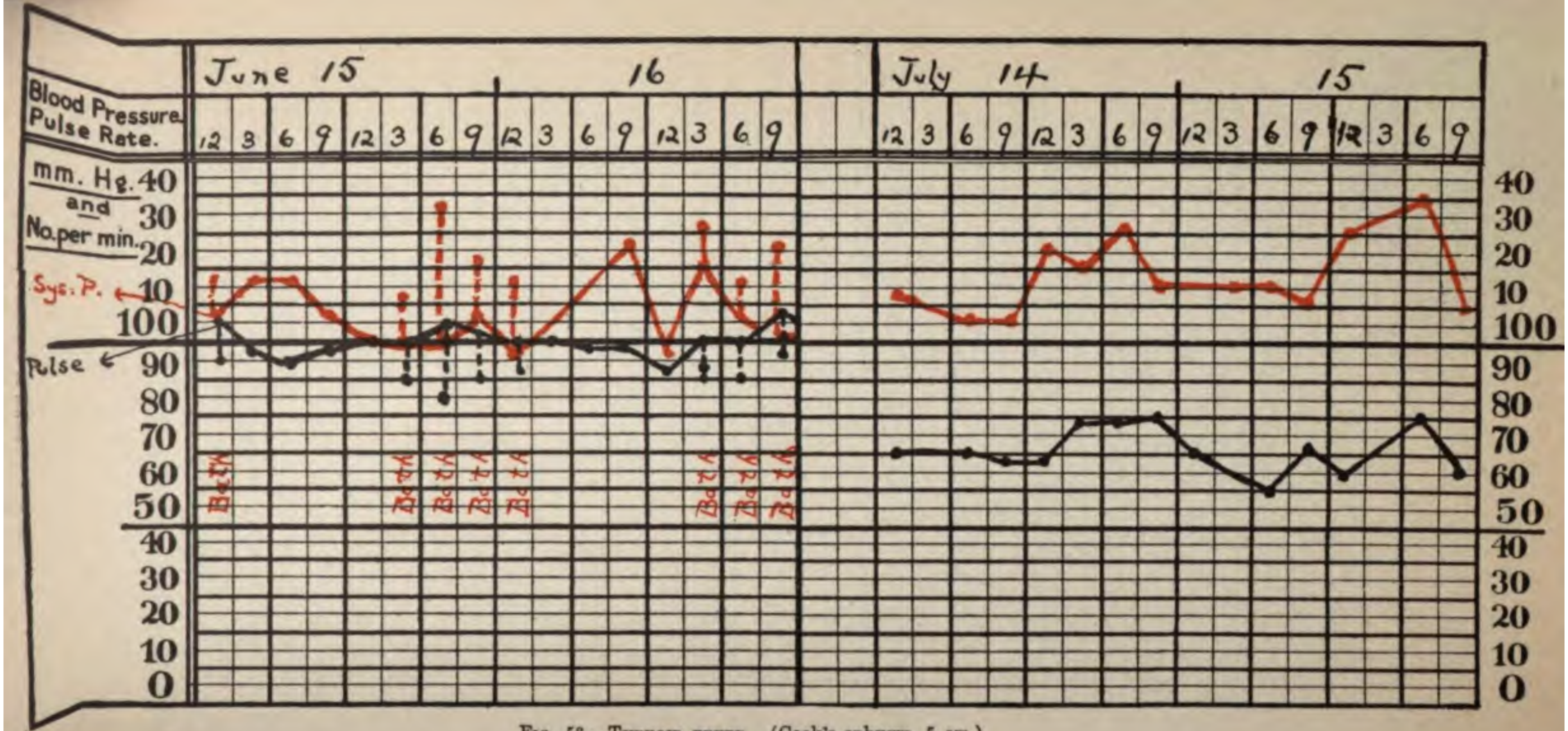


Fig. 53.—Typhoid fever. (Cook's sphygm. 5 cm.)

Portions of a chart from an uncomplicated case of moderate severity in a man of twenty-four.
June 15th and 16th were typical days early in the third week, showing the rise in pressure from bed-baths at 70° F. for fifteen minutes. No other treatment. Temperature range, 102°–104°.
July 14th and 15th were the fifteenth and sixteenth days of convalescence, when the patient first sat up out of bed.

25 mm., are of no serious import. Crile's figures are of particular interest.

In 115 cases the highest pressure = 138 mm. (R. R. 5 cm.).
" lowest " = 74 mm.
" mean " = 104 mm.

The mean pressure of all cases, by weeks of the disease, was as follows: first week, 115 mm.; second, 106 mm.; third, 102 mm.; fourth, 96 mm.; fifth, 98 mm. Nothing could better illustrate the gradual development of hypotension.

Hensen, Neisser, and Hayaski also report on smaller groups of cases.

a. Hæmorrhage and Collapse.—A slowly progressive fall in pressure is evidence of increasing weakness of the vaso-motor centres, and of the danger of impending collapse. It calls,

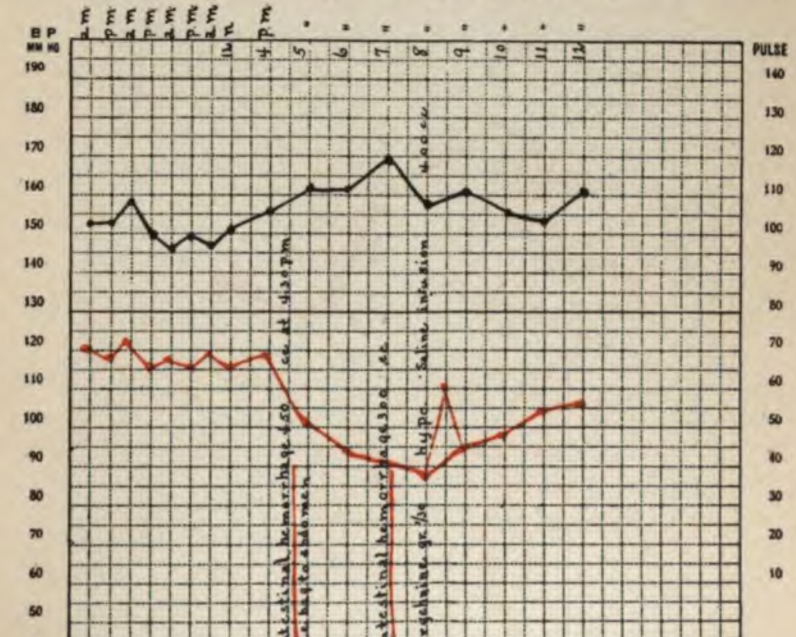


FIG. 54.—TYPHOID FEVER—INTESTINAL HÆMORRHAGE. (Cook's sphygm. 5 cm.)

Blood-pressure level broken by a sharp fall with the first hæmorrhage, with no precedent rise in tension—distinction from intestinal perforation. The fall continues with a second hæmorrhage.

Gradual return of blood-pressure after the cessation of hæmorrhage. The physiological return aided, though the curve is not materially altered, by a small dose (gr. 1/30) of strychnin and a small saline infusion. (From Cook and Briggs, Chart No. XXIII.)

therefore, for stimulant medication. A rapid fall suggests hæmorrhage. Neither Cook and Briggs, nor Alezais and Francois, failed to observe this in any case. All other complications are attended by some fall in pressure, except pneumonia and perforative peritonitis.

b. Perforation.—In the latter condition Crile, and Briggs, have both found a sharp rise marking its onset. The number of cases does not suffice for absolute statements, but the similar heightening of tension in peritonitis from other causes, which Crile found invariable in 20 surgical patients, makes it probable

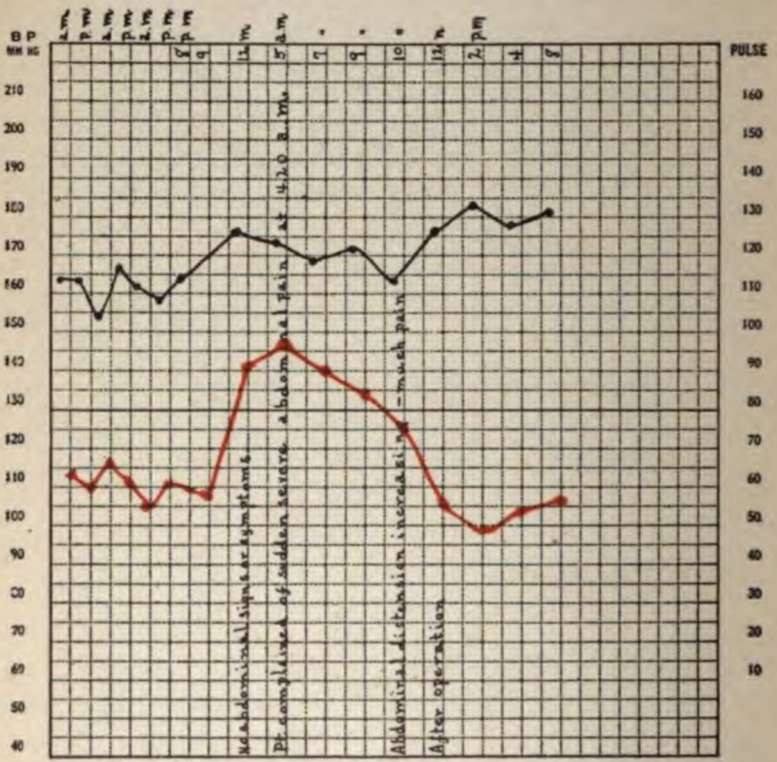


FIG. 55.—TYPHOID FEVER—INTESTINAL PERFORATION. (Cook's sphygm. 5 cm.)

Level curve of blood-pressure for days before the onset of the complication.
Sharp rise, amounting to 36 mm. Hg. in blood-pressure, over four hours before the onset of symptoms pointing to the abdomen, and at a time when the abdomen was soft and tender, and not distended.
Tendency of the blood-pressure to return to a low level as the toxæmia deepened. Slight post-operative depression. (From Cook and Briggs, Chart No. XXII.)

that a relative hypertension will be the usual result of perforation. In one of Crile's patients the pressure rose from 116 mm. to 165 mm. in two hours, and, in another, from 116 mm. to 190 mm. in four hours. In two others, without previous record, 165 mm. and 208 mm. were the figures, a striking contrast to his highest reading of 138 mm. in uncomplicated typhoid. In a fifth case, the elevation was only from 84 mm. to 110 mm.

Since the differential diagnosis of perforation is always difficult, and the other accidents which come in question, concealed hæmorrhage and acute collapse, produce a fall, this sharp elevation of pressure may often be of inestimable value.

From the other aspect of the problem Briggs reports one case, with clinical symptoms pointing to perforation, but no pressure rise. Operation was undertaken, but the perforation was not present. Full reports of all such cases should be made, that the value of the blood-pressure chart, both as positive and negative evidence of perforation, may be established. A later fall of pressure, as a terminal event in the subsequent peritonitis, must be distinguished from the initial hypertension, which can only be detected in continuous records. It is of no use to apply the sphygmomanometer after abdominal symptoms occur, without knowledge of the previous trend of pressure.

c. Therapeutics.—The bath treatment of typhoid fever seems to have as distinct an effect on the blood-pressure curve, as on temperature and pulse. When it acts favorably, a considerable rise is produced, as shown in Fig. 53. Mercandino found a more uniform rise from it than from any stimulant drug. I cannot but feel that this hypertensive effect is evidence of an action on the vaso-motor system, which is of more importance than the reduction of temperature, and that the sphygmomanometer may give indications for the employment or abandonment of this method of treatment in particular cases. Cook and Briggs have found strychnin and digitalin the most useful drugs for combatting collapse and hypotension. Where urgent need exists, they give as much as one-tenth of a grain of either. Digitalin acted the more quickly. A combination of both seemed most generally useful. Mercandino, on the other hand, and Schüle, as is the case with continental practitioners in general, prefer camphor and caffein to all others. This accords with Pässler's experimental findings in collapse, strychnin having acted on blood-pressure only in toxic dose, digitalis temporarily through strengthening the heart, and caffein and camphor causing a considerable rise. It is to be hoped that clinicians generally will study the action of drugs on blood-pressure in acute disease. The subject, like all clinical therapeutics, is involved in all manner of contradictions to-day. It is possible that some remedial agents are of use in disease, which, neither in normal men, nor in animals, can be proved to have definite physiological effects. The stimulants, however, cannot fall into such a category, and we should not employ as cardio-vascular stimulants, drugs, whose effects on blood-pressure cannot be demonstrated. Empiricism is still essential to the practice of medicine, because the scientific study of disease has covered but a small portion of its field; but let us be honest empirics, when we must, and stop hiding our

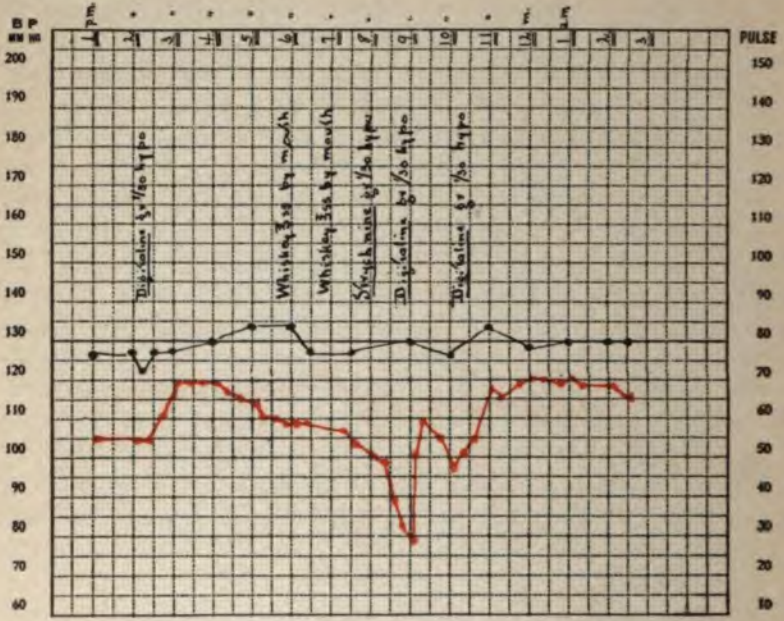


FIG. 56.—TYPHOID FEVER WITH TOXÆMIC FEATURES AND HYPOTENSION. (Cook's sphygm. 5 cm.)

Rise in blood-pressure following each of several doses of digitalin hypodermically. Alcohol by mouth and strychnin hypodermically, without any beneficial effect on the blood-pressure, even during a period of acute depression (threatened collapse). (From Cook and Briggs, Chart No. XXV.)

ignorance behind the mask of scientific terminology. Least of all so-called stimulants does alcohol deserve the name. As in animals and normal men it is without influence on the blood-pressure or the force of the heart, so in disease the same holds true. Cook and Briggs, Cabot, Mercandino, Schüle, and Swientochowski, have all corroborated this by sphygmomanometric studies. This does not negative any value of alcohol as a food, or for effect on the nervous system or blood-distribution, in fevers ; but it does teach us not to rely upon it as a means of fighting

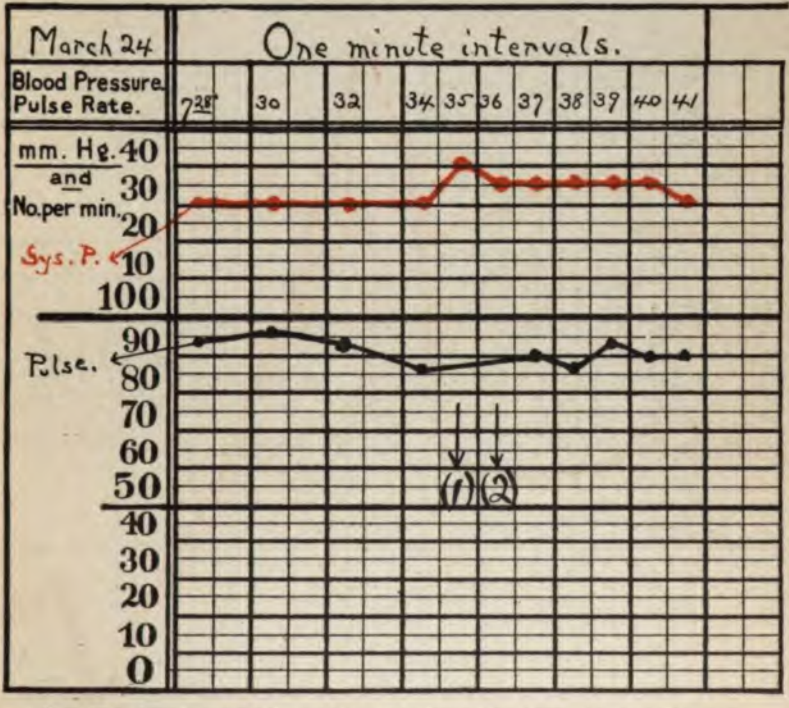


FIG. 57.—ABSENCE OF ANY RISE IN BLOOD-PRESSURE AFTER THE SUBCUTANEOUS INJECTION OF ADRENALIN. (Author's sphygm. 12 cm.)

(1) Puncture, with rise from peripheral irritation.
(2) Injection of adrenalin chloride (1 in 1,000), ♏x, which missed the vein.

cardiac or vaso-motor collapse. In fact some danger seems possible from its use under such circumstances. Ether, as might be expected, follows the same rule. The wonderful peripheral constrictor action of adrenalin is, unfortunately, so

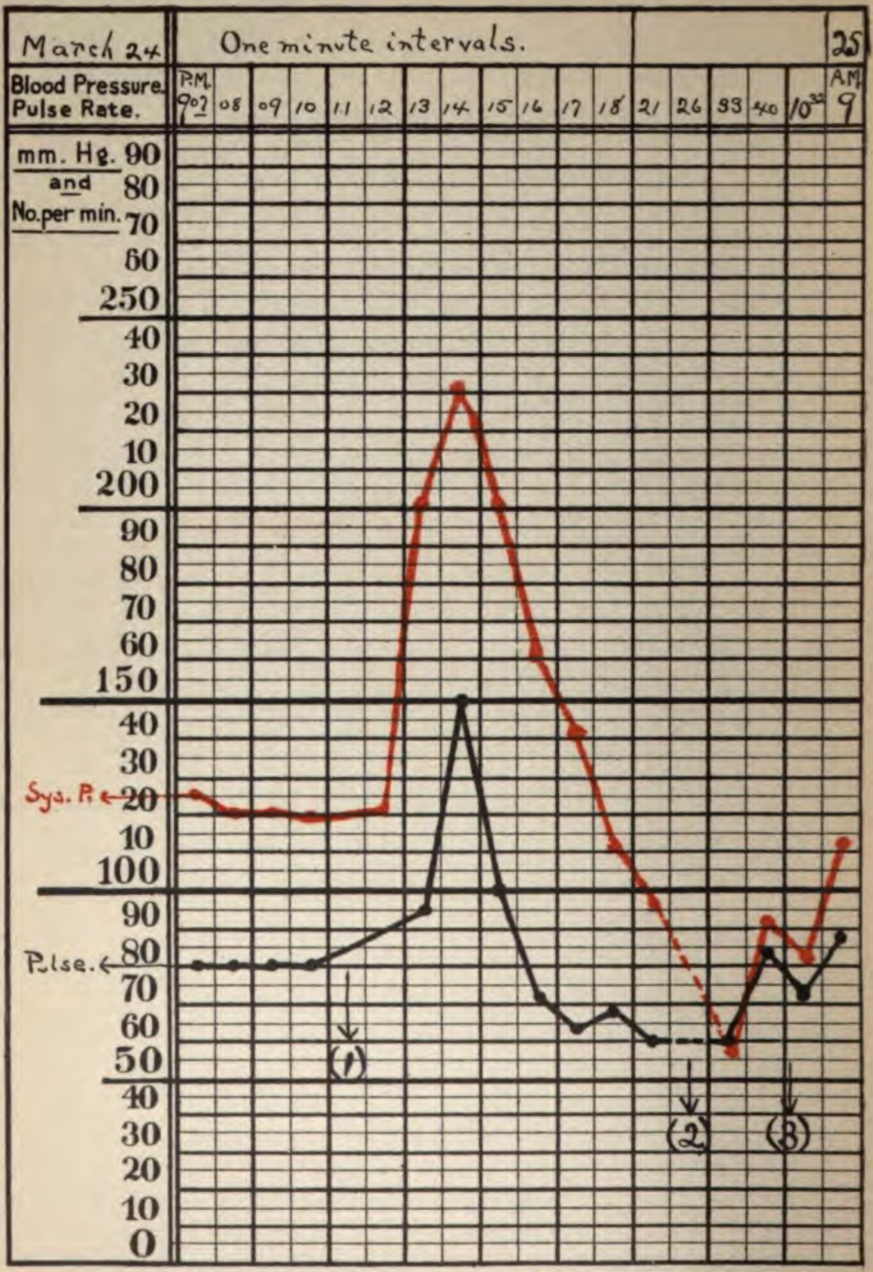

March 24
One minute intervals.
25
Blood Pressure
Pulse Rate.
P.M. 9 07
08
09
10
11
12
13
14
15
16
17
18
21
26
33
40
10 32
A.M. 9
mm. Hg. 90
and 80
No. per min. 70
60
250
40
30
20
10
200
90
80
70
60
150
40
30
Sys. P. ← 20
10
100
90
Pulse. ← 80
70
60
50
40
30
20
10
0
(1)
(2)
(3)

FIG. 58.

FIG. 58.—EXTREME RISE IN BLOOD-PRESSURE PRODUCED BY THE INTRAVENOUS INJECTION OF ADRENALIN. (Author's sphygm. 12 cm.)

(1) Adrenalin chloride (1 in 1,000), ♏xx, injected into the median basilic vein.

With the rise, which followed instantly, there was throbbing in head, excitement, substernal pain, and nausea.

(2) No pulse palpable; heart sounds very faint; patient delirious, pale.

Strychnin sulphate gr. 1/30 and atropin sulphate gr. 1/200 given hypodermically.

(3) Caffein sodium salicylate, gr. ii, given hypodermically.

In spite of the alarming secondary depression (an unusual result), there were no bad after-effects.

fleeting as to be of no practical service in acute disease. It must also be injected into the vein to act at all (see Figs. 57 and 58). Crile had no success with his pneumatic suit in a few cases. Probably coincident failure of the respiratory centre excludes help from this measure.

d. Late Effects.—Thayer has examined the systolic blood-pressure, as well as the condition of heart and arteries, in 165 persons who had formerly passed through typhoid fever in the Johns Hopkins Hospital. His figures show an average higher pressure than was obtained in a comparative series of 276 healthy persons who had not had the disease, either when arranged by decades, or for the entire group. What is much more significant, 27 had more than 180 mm. pressure, while 10 were above 200 mm. (R. R. 5 cm.). Of the normal individuals, only one woman of sixty showed a tension of 180 mm. Other infections and possible alcoholic excesses were excluded, as carefully as might be, from the list. The figures, with Thayer's other findings, seem to support his contention that cardio-vascular changes frequently follow the typhoid infection, manifesting themselves more especially in later years.

B. Pneumonia.—One finds little uniformity of results in looking over blood-pressure charts in cases of pneumonia. This holds true for one writer as against another, and for many observers in their individual experience. The explanation is evidently that pneumonia has no such constant effect on pressure as typhoid fever. Potain classes it as a disease with normal pressures, Cook and Briggs, and Gilbert and Castaigne as hypertensive, while Hayaski, Kaufmann and de Bary, and Neisser believe the pressure is usually low. Probably the arterial pressure is as variable as many of the other clinical manifestations of the disease.

Fraenkel has made regular determinations with the tonometer in all his cases for some years. He finds a subnormal pressure the rule, especially at the time of the crisis. In one-third of the cases, however, it was absent. He does not attach much value to the tonometric observations. Neisser, also with the tonometer, found a sharp fall at the crisis frequent. The instrument seems to me rather ill chosen for a disease in which peripheral cyanosis, and probable altered tonus of the smaller arteries, is so common. Giglioli reports 50 cases with the Riva-Rocci apparatus. In the favorable ones the change in pressure was slight. In severe cases he saw a fall to 80–90 mm. on the fourth or fifth day. In fatal cases there was a rapid fall, he says, with dilatation of the heart. Neither Hensen, nor Mosen could demonstrate the critical drop in pressure, while Jarotzny's lowest recorded reading was under just these circumstances. Gilbert and Castaigne, while their figures cannot be accepted on account of the instrument used (P.), yet agree well with Giglioli. They found slight hypertension during the first day or so. In favorable cases the tension never decreased materially, while in fatal cases a rapid fall to 80 mm. or less occurred. During convalescence it returned slowly to normal. They believe that a drop to 90 mm. (P.) signifies serious danger. My personal observations are not numerous enough to allow of deductions. There seems to be great daily variation in pressure. Nevertheless, the charts have been distinctly helpful as a guide to treatment, the general direction of the pressure and pulse curves, not their absolute height, being the valuable feature. A rise in pressure with fall in pulse, as improvement set in, has been conspicuous (see Fig. 33).

When one pictures the possible causes for variation in lobar pneumonia, the difference in individual reaction to the toxæmia and in extent of lung tissue involved, the motor restlessness of some patients, the urgent dyspnœa of others, and the great likelihood of an asphyxial rise of pressure where cyanosis is extreme, there is little wonder at these somewhat discordant results. Conditions are not so simple as in the pneumococcus septicæmia of Romberg and Pässler. However, in pneumonia, more than in any other disease, collapse comes without warning. If, in even a portion of the cases, a blood-pressure chart will give notice of its approach, the chart is worth while. This is the more true, because, in this disease, one may fight for even minutes with the hope that the tide will turn. I hope to make observations on the blood-pressure reaction to stimulation of the nasal mucosa, which in man, as in the rabbit, may give a much earlier indication of vaso-motor paralysis than the actual level of pressure. The treatment of lobar pneumonia is so entirely an empirical matter that I can-

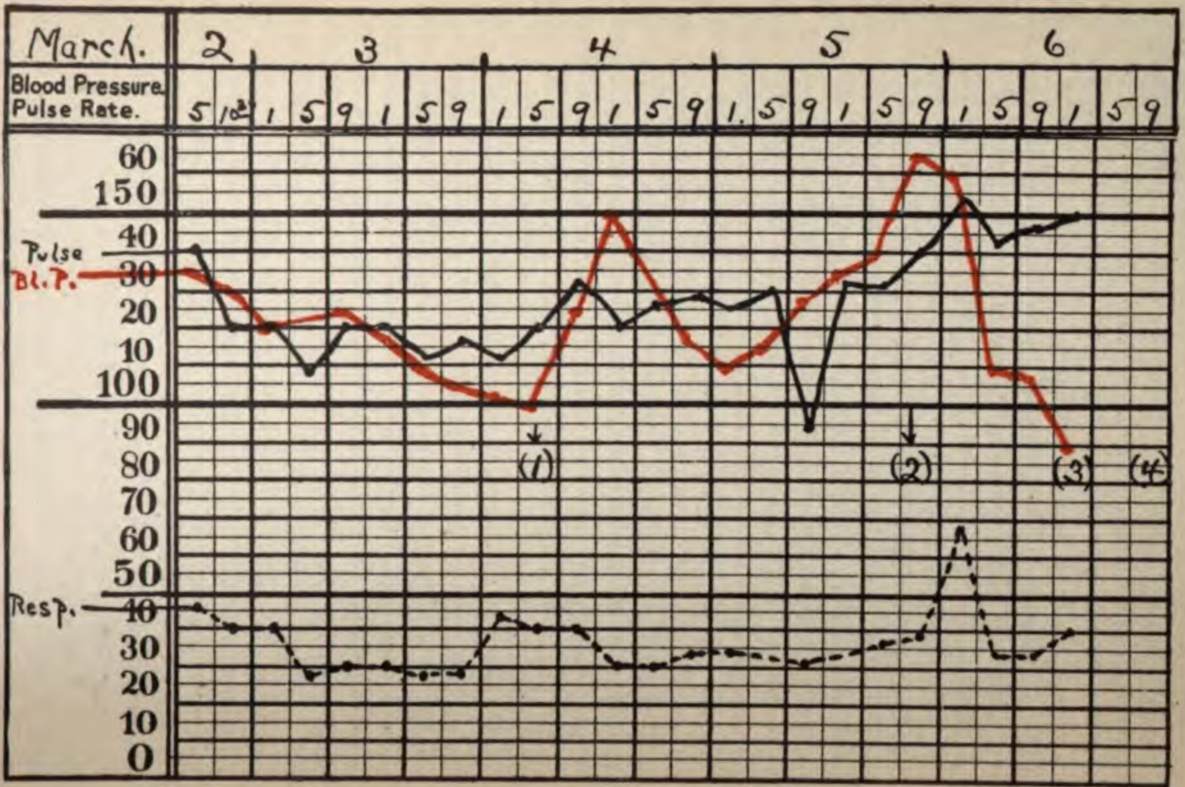

March.
2
3
4
5
6
Blood Pressure.
Pulse Rate.
5 10 1 5 9 1 5 9 1 5 9 1 5 9 1 5 9 1 5 9 1 5 9 1 5 9
60
150
40
30
20
10
100
90
80
70
60
50
40
30
20
10
0
Pulse
Bl. P.
Resp.
(1)
(2)
(3)
(4)

FIG. 59.

Fig. 59.—Fatal lobar pneumonia. (Author's sphygm. 12 cm.)

Chart from J. O'N., City Hospital, Ward 1. Autopsy showed hepatization of entire right lung and of left lower lobe.

(1) Rise in pressure as muscular twitching became marked; probably fictitious. Pulse small.

(2) Sharp rise from intravenous saline infusion.

(3) Rapid fall in pressure, with rising pulse, beginning twenty-four hours before death. No further readings possible.

(4) Died at 10.20 P. M.

not enter upon it from the standpoint of blood-pressure, more than to repeat what has already been said under typhoid concerning stimulant measures.

C. Diphtheria.—Only a few clinical studies of blood-pressure have been made in diphtheria. The most extensive, by Friedmann, was unfortunately in the days of the v. Basch sphygmomanometer. Of 15 cases in his series, in which marked hypotension came on during the first week, only two recovered. These two were both severe. His results should have further confirmation in a large number of patients. This will only be possible in a diphtheria hospital, but is most desirable. In laryngeal diphtheria, all the conditions for a true asphyxial elevation exist. This was seen by Shaw and carefully studied by Hensen. One girl, for example, had a pressure of 130 mm. (R. R. 5 cm.) and no respiratory variation. She became suddenly cyanotic, with stridulous breathing, and the pressure rose to 155 mm., with each inspiration sinking to 140 mm. Soon the breathing became free again, and the pressure dropped at once to 130 mm. This blood-pressure representation of the paradoxical pulse he found in a number of cases of laryngeal diphtheria.

D. Other Acute Infections (Malaria; acute rheumatism; septic conditions; influenza).—In none of the other acute febrile diseases is there anything very definite about the state of the blood-pressure. The older writers, with the v. Basch instrument, studied malaria with results in accordance with their particular theory as to the relation of temperature to blood-pressure. Norris recently could make out but little variation, in spite of a great temperature range. Potain sets down acute articular rheumatism as a hypotensive disease, but Carter, Norris, and Hensen found no marked change. The latter, like Giglioli, Hayaski, and Neisser, found that in most febrile diseases the pressure after a time tended to become subnormal. In none of them has its observation been shown to have any great use. Hensen studied a number of septic conditions, and Federn lays great stress on the subnormal pressure in epidemic influenza, which he thinks due to heart weakness. He also says that persons with previous hypertension stand the disease badly. Acute peritonitis will be considered with surgical conditions in Chapter IX.

E. Acute Infections in Childhood.—Cook has made important practical studies in children sick with acute diarrhœal diseases, pneumonia, pertussis, and tuberculosis, as well as in marasmus and rickets, which scarcely belong here. In all these conditions, falling blood-pressure was found almost uniformly as an early sign of a change for the worse, a rapid drop indicating serious collapse. The decline in pressure was often the first evidence of trouble, and, in his experience, constituted the safest guide to the employment of stimulant measures. During the first year, 60 mm. usually marked the danger line, when treatment was called for; in other children, 80 mm. The general course of the chart, however, was more valuable than any single actual reading. In collapse, pressure might fall 30 mm. within an hour or so.

a. Therapeutics.—The drugs which gave the best results, judged by their pressor effects, were digitalin and strychnin, from gr. $\frac{1}{400}$ to gr. $\frac{1}{200}$, especially in sudden collapse. Alcohol in single doses was without constant effect, but repeated small doses sometimes caused a gradual augmentation of tension. Saline infusion, as a stimulant, was worthless, though frequently acting beneficially on the general condition, perhaps through the elimination of toxins. In collapse with cyanosis, a hot mustard bath occasionally worked well. A short-lived response to treatment, with renewed fall, argued a bad prognosis.

3. CHRONIC INFECTIOUS DISEASES

A. Tuberculosis.—The earliest studies by Marfan led him to believe that low tension is one of the most constant symptoms of phthisis, appearing even in the incipient stage. Of a hundred patients, only three showed normal pressure, and these were old people with arterio-sclerosis. His unqualified statement may find some explanation in the fact that Potain's sphygmomanometer would be likely to give lower readings in thin persons.

Since that time, systematic observations on the blood-pressure of consumptives have been published from several European sanitoria, by Burckhardt, John, and Naumann, all with the Gärtner tonometer. A number of isolated measurements of less value are given by Potain (P.), Jarotzny (H. & B.), Hensen (R. R.), and Hayaski (R. R. & G.). All agree that hypotension is the rule in the more advanced stages, running roughly parallel with the impairment of general bodily vigor.

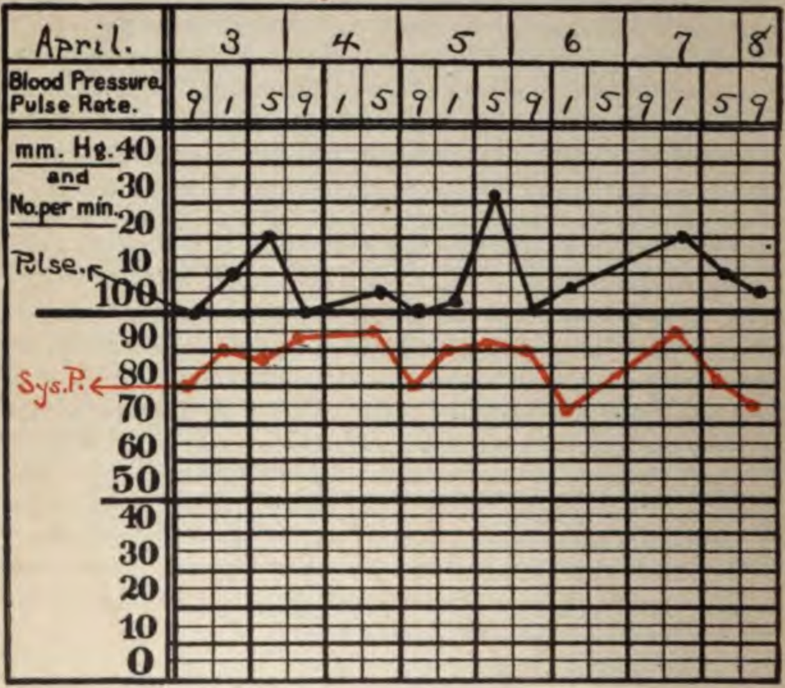


FIG. 60.—MARKED HYPOTENSION IN ACUTE TUBERCULOSIS. (Author's sphygm. 12 cm.)

Portion of chart from a case of acute phthisis, with terminal general miliary tuberculosis (autopsy), who died of hæmorrhage, April 13th, ten weeks from onset of disease. F. P., male, aged twenty-nine, City Hospital.

Burckhardt, in rather a small series, found the pressure regularly diminished early in the second stage of the disease, and, in advanced phthisis, rapid pulse and subnormal tension were invariable. Light exercise produced a rise in some patients and a fall in others, never of great extent.

Naumann studied 100 cases, from which all who had fever, arterio-sclerosis, heart lesions, pleural adhesions, or albumin or sugar in the urine, were carefully excluded. They were, therefore, patients with chronic, practically inactive and uncomplicated pulmonary tuberculosis. In this they differ from Burckhardt's patients, most of whom had an active febrile process. Of these 100 patients,

in 69 the blood-pressure was over 130 mm. (G.),
in 13 " " " " 115 to 130 mm. (G.),
in 18 " " " " under 115 mm. (G.).

Naumann considers these, high, normal, and subnormal values. As to incidence with the extent of the pulmonary lesion, of the 69 patients with over 130 mm., there were,

in 1st stage (Turban), 28,
in 2d " " 22,
in 3d " " 19.

Evidently the area of lung involved is without influence. In order to reconcile these results with the apparently uniform

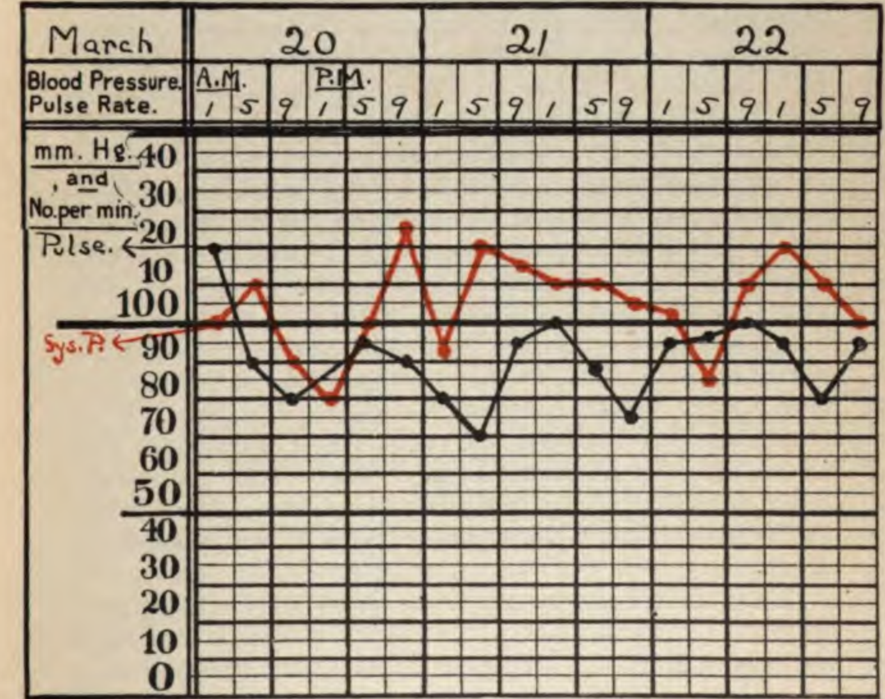


FIG. 61.—CHRONIC PULMONARY TUBERCULOSIS WITH SLIGHT HYPOTENSION. (Author's sphygm. 12 cm.)

Portion of chart from J. L., male, aged sixty-three, City Hospital. Advanced process with fever. Patient died April 22d, with marked hypotension.

This chart represents the usual course of blood-pressure in such cases until near the end, when hypotension becomes marked.

hypotension of the advanced febrile cases, one must suppose that chronic toxæmia is its probable cause. Naumann believes that arterial tension is valuable in prognosis, as showing to some extent the constitutional disturbance, which is unquestionably a better guide to the future than the local signs.

For treatment, it is hard to see that a knowledge of the blood-pressure adds much, save that a continuously low level is additional argument against physical exertion. The general causes of low pressure, the toxæmia and malnutrition, with their results in heart and skeletal muscles, are the proper objects of therapeutic attack, not the circulatory system.

a. Hæmoptysis.—Burckhardt quotes a case, which he tries to bring in line with Hensen's observations on hæmoptysis from aneurism, and hæmatemesis, as showing a rise in pressure after hæmoptysis. From his own figures, 8.5 cm. before, 7.5 cm. the next morning, 10 cm. the second day, there was a primary fall. His determinations were at too great an interval to be valuable. If the pressure does rise during a hæmorrhage, as Hensen showed it might, I am inclined to look for the reason in the attendant psychical excitement. Certainly in surgical operations and in typhoid fever, where the mental factor is absent, a fall in pressure is practically invariable. Schüle reports hypotension after hæmoptysis, as low as 60 mm. (G.), and Cook and Briggs show a fall from 125 to 70 mm., due to a hæmorrhage of 1,100 c.c. Naumann attempts to correlate the higher tension in 69 of his cases with the tendency to hæmoptysis. Of 51 patients who had bleeding at some time, 86.2 per cent. showed a pressure above 130 mm. Of these 44 with hypertension (?) and hæmoptysis, 24 were in the early stage. From this he concludes that most hæmoptyses occur in the early cases with high blood-pressure. These results need confirmation.

B. Syphilis.—It is not my purpose to discuss in any way the relation of a past syphilitic infection to those late anatomical changes in heart and vessels, which have already been considered. In the early stages of syphilis, however, when the process clinically resembles a subacute or chronic infectious disease, circulatory disturbances are frequent. These usually show as minor grades of cardiac insufficiency, with the development of a mitral systolic murmur. Grassmann has studied the blood-pressure in 64 such cases, making repeated observations in 61, with the v. Basch sphygmomanometer. In nearly all of them he found hypotension more or less marked. Eighty per cent. had a pressure below 80 mm. (right temporal artery) at the beginning of treatment.

The behavior of the blood-pressure during the further course of the disease varied. Twelve cases showed no change, while 48 had considerable fluctuations. Of these, the majority presented a further fall in pressure averaging 15 to 20 mm., developing a little time after the institution of mercurial treatment. As a rule the hæmoglobin was simultaneously reduced. In 37 patients with subnormal initial tension, 7 showed a further lowering; 9, a sinking, followed by a rising pressure; 17, a rise; and 4, a rise with subsequent fall. For the entire 61 patients, the results of treatment were as follows: stationary pressure, 13 cases; an increase, temporary or sustained, 22 cases; a decrease, 26 cases.

Grassmann believes that the relatively small number of patients in whom the blood-pressure remains normal during early syphilis are an indication of the frequency with which damage to the heart, of greater or less extent, occurs at this stage. He considers the hypotension a sign of relative insufficiency of the left ventricle. Unfortunately, improvement in the cardiac condition, as shown by the disappearance of relative mitral insufficiency, sometimes coincided with an increase, sometimes with a reduction of tension.

4. CHRONIC DISEASES ASSOCIATED WITH IMPAIRED NUTRITION

For convenience it is necessary to include under this heading a somewhat heterogeneous group. The blood-pressure findings are of no special importance in any of them, except as pointing to unnoticed complications from the side of kidneys, heart, or arteries.

A. Diabetes.—Potain laid much stress on diabetes as a cause of the extreme grade of hypertension. His dictum seems to have been accepted by many other observers without verification. Certainly there exist no satisfactory statistics on the subject, and even isolated observations are hard to find. Crummer, for instance, noted high pressure. On the other hand

Hensen, in his Table III, headed Cachexia and Marasmus, includes two cases of diabetes with slight hypotension. I have seen both high and low blood-pressure in diabetes, the latter in severe cases with marked emaciation and diacetic acid in the urine; the former especially in the milder forms in stout elderly people, where chronic nephritis or arterio-sclerosis existed. I believe that the disease, of itself, is without influence on arterial pressure; that the occurrence of chronic nephritis, or arterio- or angio-sclerosis, as a complication, explains the hypertension; and that the resultant emaciation, and brown atrophy or fatty change in the heart, cause the hypotension in severe cases. Tabulated observations on a large number of cases, with careful notes of the complicating conditions, will settle this still open question.

B. The Anæmias.—The condition of the arterial pressure in anæmia has been a matter for considerable discussion, especially since many authors, who use the sphygmograph as an instrument of precision, have insisted on the occurrence of prolonged tension. Almost all the measurements of blood-pressure in anæmic states are against this view. As a matter of fact, the hypotension of cachectic states is present in most of the secondary anæmias, and the causes of hypertension may coexist with others, chronic nephritis especially. Chlorosis is the form in which the effect of hæmoglobin deficiency per se should be demonstrated. The general consensus of opinion seems to be that some reduction of pressure exists in chlorotics, but it is neither marked nor proportional, in any but the roughest way, to the grade of anæmia present. I cannot see that blood-pressure measurements add anything valuable to our clinical knowledge of the anæmias. They are useful mainly as indicating possible complications, or primary causative conditions in the case of the secondary varieties.

v. Basch originally taught that anæmia is associated with low arterial pressure. Bihler studied the relation of pressure and hæmoglobin in 50 chlorotic girls. The average hæmoglobin on admission was 58 per cent., systolic pressure 77 mm. (v. B.); on discharge, 94 per cent. and 86 mm. This difference lies within the possible error of the method, but, in 50 cases, may be accorded some value. He believed it was related to the cardiac weakness, so common in chlorosis. Potain considered chlorosis a disease with normal pressures. Carter, and Jarotzny, both found low tension (H. & B.). With the tonometer, Schüle, and Crummer saw low pressures, the latter some very low readings. Shaw, however, in children, said tension was slightly increased. With the Riva-Rocci, Giglioli examined 30 anæmias of different origins, all having hypotension, the lowest, 70 mm., a pernicious anæmia. Cook and Briggs believe hypotension is the rule, but dependent more on the state of general nutrition than on the blood condition. Hensen saw little change, but examined very few cases.

C. Cachectic States.—The extreme impairment of nutrition which we call cachexia, is unquestionably associated with hypotension in the majority of cases. Both v. Basch and Potain were convinced of this, and, with the newer instruments, Cook and Briggs (R. R.), and Schüle (G.), among others, have substantiated it. Cases of cancer, especially of the digestive system, of advanced tuberculosis, of syphilitic cachexia, etc., have a blood-pressure more or less in accord with their general state of nutrition (see Fig. 60). The lowest reading I have ever seen in office practice was in a patient with cancer of the œsophagus, whose strength was still fair. Systolic pressure was 80 mm., diastolic, 55 mm. (J. 12 cm.). Marked hypotension, however, that is, below 80 mm. (5 cm.), 70 mm. (12 cm.), does not occur except as a terminal event.

Hensen calls attention to a fact, which I believe is too often lost sight of, to wit, that even profound malnutrition may be associated with a comparatively high arterial pressure. He cites one case of stenosis of the œsophagus with arteriosclerosis, who showed a systolic pressure of 183 mm. (R. R. 5 cm.). One must always remember that cardio-vascular disease with high tension may coexist in these cases, though probably the pressure does not reach the figures it would, were the general health better.

a. Addison's Disease.—I call attention to two observations of my own in this connection, because it is usually believed that extreme hypotension goes with the disease. One was a woman who was still about, but with a typical clinical picture of Addison's and a diastolic pressure of 90 mm. (H. & B.); the so-called mean pressure was 105 mm. The other, a man in my service at the City Hospital, was in the last stages of emaciation and asthenia, and showed at autopsy typical fibrosis and caseation of both suprarenals. A number of determinations, up to within about two weeks of his death, showed a systolic pressure of 140 mm. in one arm. In the other, the radial was exceedingly small and systolic pressure was about 100 mm.

5. MISCELLANEOUS

Only conditions which are important, or in which we have actual knowledge of the blood-pressure, are included here. The results of notoriously inaccurate methods or of a priori reasoning, which one meets in the literature, will not be considered.

A. Lead Poisoning.—Chronic lead intoxication, as is well known, frequently produces changes in arteries and kidneys, and thus permanent hypertension as a secondary effect. Apart from such anatomical alterations, however, lead poisoning, as evidenced by the typical colic, almost always goes with moderately high blood-pressure. Christeller, Hensen (8 out of 10 cases), Norris (8 out of 9 cases), and Thaussig (3 cases), report this as the rule. Hensen, and Norris, in isolated cases, especially with intoxications of short duration, failed to obtain this hypertension ; and Gumprecht, and Butterman note normal pressures for a case or so.

Borgen made the most careful study of the condition, in a series of 19 patients, unfortunately with the v. Basch sphygmomanometer. From the figures obtained he divides the clinical course of lead colic, as seen after admission to a hospital, into four stages.

1. Stage of rising pressure, duration indefinite, twelve hours to several days.
2. Stage of high pressure, one to four days, or longer. The pressure remains equally high during and between the attacks of pain, which have already begun in the first stage.
3. Stage of falling pressure. Pain and tenderness disappear rapidly, appetite and bowels become normal. Pressure sinks slowly to normal during a day or two.
4. Stage of subnormal pressure. Not present in all patients. On discharge, pressure usually above the average, but may be below.

Borgen found no relation between the actual paroxysms of pain and the hypertension, which was continuous. Morphin seemed to reduce the tension slightly. Thaussig reports some remarkable effects from, or coincident with, treatment with iodipin, administered hypodermatically in doses of 5 c.c., increased to 15 c.c. One case, in twenty-eight days, dropped from 220 mm. (G.) to 110 mm., with improvement in all symptoms; another in ten days, from 200 mm. to 90 mm.; the third, in eight days, from 160 mm. to 80 mm.

Pal reports a case of unusual interest, in which the amaurosis, occasionally seen in lead colic, seemed to stand in direct relation to the hypertension.

The patient, a man of forty-one, was admitted to the clinic on the sixth day of a typical attack, having passed through a similar one three years previously. On admission, pressure was 160 mm. (G.), pulse 78. Eyesight and pupillary reactions were normal. The abdomen was retracted and tender, and the patient complained of considerable pain. Neither fæces nor wind were passed. Urine contained neither albumin nor sugar.

On the next morning the patient noticed impairment of vision, which increased to complete amaurosis during the day. Headache occurred. The pupil was normal, papilla pale, vessels tortuous, otherwise a normal ophthalmoscopic picture. Tonometer reading 170 mm. in the morning, 180 mm. in the evening. He was ordered 5 grams of potassium iodide per day. The subsequent day the patient could distinguish between light and darkness. Pain was less, and a good movement followed the use of senna. Pressure, A. M. 165 mm., P. M. 160 mm. A day later he could count fingers, and pain had disappeared. Pressure 145 mm. In one day more vision was restored, and the tension had fallen to 125–120 mm. Recovery was rapid and, on the day of discharge, his pressure was only 75 mm.

Altogether it seems proven that lead must be kept in mind as a possible cause of marked hypertension, in cases without demonstrable nephritis or arterio-sclerosis. While it is not invariable, the other diagnostic features, blue line and granular degeneration of the red blood cells, are also lacking in some patients, so that the sphygmomanometer may assist in the decision of an obscure case.

B. Gout.—Clinicians have been accustomed to associate high pulse tension and gout, yet no sphygmomanometric observations have yet been reported which can be accepted. Jourdin and Fischer state that in gout, which is a hypertensive disease, a fall in pressure presages an acute attack, but do not quote their authority. It is to be hoped that we may have some objective testimony soon, for it would be of distinct theoretical importance in connection with the etiological factors in arterial disease, and the relation of the latter, and of nephritis, to gout.

C. Emphysema; Chronic Bronchitis; Asthma.—Very little attention has been paid to these conditions, in which dyspnœa is so marked a feature. Hensen alone has more than a passing reference to them, Carter, and Norris merely noting a case or so with fairly normal pressure. Hensen regularly found comparatively high tension (135 to 180 mm. R. R. 5 cm.), in out-patients with emphysema, asthma, and kyphoscoliosis. This has been my experience in office practice, where no clinical evidence of nephritis or arterial change existed. In many of my cases I am inclined to think of latent angio-sclerosis as the probable cause, since arterial disease and emphysema are frequent companions at the autopsy table. Hensen thought the dyspnœa was the causative factor; but the arterial pressure may be permanently high, while dyspnœa only follows exertion. One patient, with advanced emphysema, following many years of chronic bronchitis with asthma, had a systolic pressure of 140 mm., diastolic 110 mm. (J. 12 cm.), in spite of a dilated right heart with tricuspid systolic murmur, and œdema of the legs. Such cases show well the futility of judging the condition of the heart from the height of blood-pressure.

In asthma, Hensen repeatedly observed elevations in tension of 20 mm. and more at the approach of an attack. Also, during the paroxysm, marked inspiratory sinking of pressure. Nervous influences, as well as the dyspnœa, must be in mind in such a distressing condition as spasmodic asthma.

D. Pleural and Peritoneal Effusions.—A small number of blood-pressure determinations, before and after the removal of pleural and peritoneal effusions, show a constant fall as the result of the procedure. Hensen, Kapsammer, and Norris report cases. The effect is much more marked in the case of large pleural than of peritoneal collections, and evidently indicates that the presence of the fluid causes functional hypertension.

Quirin has examined the intra-abdominal pressure during the drainage of ascitic fluid, and found positive pressures at the moment of puncture between 14 and 28 mm. Hg. With the removal of the fluid, this sank gradually, 4 mm. being the lowest and 14 mm. the highest figures at the end of paracentesis. This fall in intra-abdominal pressure of 10 to 14 mm. corresponds closely with the reduction in systolic arterial tension reported during the same procedure, usually only 5 to 9 mm. In the light of Quirin's animal experiments (see page 15), a simple mechanical increase in peripheral resistance obviously suffices to explain this slight hypertension. Cook and Briggs note a larger reduction on tapping an ascitic accumulation.

With the aspiration of large pleural accumulations, much greater differences occur. Hensen's results, in 7 cases, show 14 mm. as the least, 50 mm. as the greatest reduction in systolic pressure. The mechanical factor is inadequate to explain such considerable hypertension, and we must think either of reflex vaso-constriction or, as Hensen suggests, of a mild asphyxial effect. Both may occur. The only specific references to pleurisy with effusion, which ignore this hypertensive effect, are from the French observations with Potain's sphygmomanometer, or still more inaccurate instruments. Jourdin and Fischer go so far astray as to give hypotension as an indication for aspiration.

The chief practical value in knowing that pleural effusions tend to raise arterial pressure considerably is, that hydrothorax frequently complicates nephritis and cardiac disease. One must not refer a lower tension after aspiration to the other therapeutic measures; nor, in an obscure case of cardiac disease with albuminuria and fluid in the chest, think that hypertension proves the existence of true chronic nephritis.

E. Hæmorrhage.—Apart from typhoid fever, in which hæmorrhage seems regularly accompanied by hypotension, bleeding is seen by the internal physician most commonly as hæmoptysis or epistaxis, then from the rectum, from gastric or duodenal ulcers, and occasionally from the rupture of an aneurism. Hensen reports some seemingly paradoxical findings in a case of aneurism, which perforated the left bronchus and gave rise to repeated hæmoptysis, and in gastric and pulmonary hæmorrhages, 11 cases in all. In most of these the systolic tension was increased immediately after copious bleeding, sometimes 20 to 25 mm. above its previous level. Later there was a gradual fall, never to real subnormal figures, with subsequent return to the original reading after a few days.

While the physiologists have taught us not to expect marked or permanent effects on blood-pressure from loss of blood (see page 26), still an initial rise does not tally either with animal experiment, surgical observation, or the findings in typhoid fever. Two possible causes for this difference in the two groups of conditions are evident to me. One is the great mental excitement which attends the knowledge of bleeding on the part of the patient, and which to me seems the likeliest source of the initial rise in pressure. Neither in anæsthetized animals or men, or in typhoid, does mental perturbation occur. The other cause is, perhaps, the state of the vaso-motor system, which in operations and in severe infections has not its usual power of response, and might allow a greater fall in pressure than would occur under more normal circumstances. A large number of cases must be studied, with careful noting of the psychical, as well as the physical results of the hæmorrhage, before we shall be in a position to understand its effects on blood-pressure. Records of systolic and diastolic pressures will have the greatest value.

LITERATURE

Alezais et Francois. La tension artérielle dans la fievre typhoide. Rev. de Med., 1899, vol. xix, p. 88.

v. Basch, S. Einige Ergebnisse der Blutdruckmessung an Gesunden und Kranken. Zeitschr. f. klin. Med., 1881, vol. iii, p. 502.

Bihler. Ueber das Verhalten des Blutdrucks bei Chlorotischen und über die bei denselben vorkommenden Störungen am Herzen. Deutsch. Arch. f. klin. Med., 1894, vol. lii, p. 281.

Borgen, Thomas. Blutdruckbestimmungen bei Bleikolik. Deutsch. Arch. f klin. Med., 1895, vol. lvi, p. 248.

Briggs, J. B. A Note on the Association of a Rise in Systolic Blood-pressure with the Onset of Perforative Peritonitis in Typhoid Fever. Boston Med. and Surg. Jour., 1903, vol. cxlix, p. 343.

Burckhardt, M. Untersuchungen über Blutdruck und Puls bei Tuberkulösen in Davos. Deutsch. Arch. f. klin. Med., 1901, vol. lxx, p. 236.

Butterman. Einige Beobachtungen über das Verhalten des Blutdrucks bei Kranken. Deutsch. Arch. f. klin. Med., 1902, vol. lxxiv, p. 1.

Cabot, R. C. Studies on the Action of Alcohol in Disease, especially upon the Circulation. Med. News, 1903, vol. lxxxiii, p. 145.

Christeller. Ueber Blutdruckmessungen an Menschen unter pathologischen Verhältnissen. Zeitschr. f. klin. Med., 1881, vol. iii, p. 33.

Cook, H. W. The Clinical Value of Blood-pressure Determinations as a Guide to Stimulation in Sick Children. Am. Jour. of the Med. Sci., 1903, vol. cxxv, p. 433.

Cook, H. W., and Briggs, J. B. Clinical Observations on Blood-pressure. Johns Hopkins Hosp. Rep., 1903, vol. xi, p. 451.

Crile, G. W. Diagnostic Value of Blood-pressure Determinations in the Diagnosis of Typhoid Perforations. Jour. of the Amer. Med. Asso., 1903, vol. xl, p. 1292.

Crummer, Le Roy. On the Use of Gärtner's Tonometer. Med. Herald, St. Joseph, Mo., 1902, vol. xxi, p. 46.

Federn, S. Ueber Influenza. Wien. med. Wochenschr., 1901, vol. li, p. 1162.

Friedmann, J. H. Blutdruckmessungen bei Diphtherie. Jahrb. f. Kinderheilkunde, 1893, vol. xxxvi, p. 50.

Giglioli, G. Y. Alcune critiche e alcune richerche di sfigmomanometria clinica. Riv. crit. di clin. Med., 1900, vol. i, pp. 625 and 641.

Gilbert, A., et Castaigne, J. La tension artérielle dans les pneumonies. Séan. et Mem. de la Soc. de Biol., 1899, vol. i, p. 633.

Grassmann, K. Klinische Untersuchungen an den Kreislaufsorganen im Frühstadium der Syphilis. Deutsch. Arch. f. klin. Med., 1900, vol. lxix, p. 264. (See pp. 281–289.)

Gumprecht. Experimentelle und klinische Prüfung des Riva-Rocci'schen Sphygmomanometers. Zeitschr. f. klin. Med., 1900, vol. xxxix, p. 377.

Hayaski, T. Vergleichende Blutdruckmessungen an Gesunden und Kranken mit den Apparaten von Gärtner, Riva-Rocci und Frey. Inaug. Dissert., Erlangen, 1901.

Hensen, H. Beiträge zur Physiologie und Pathologie des Blutdrucks. Deutsch. Arch. f. klin. Med., 1900, vol. lxvii, p. 438.

Jarotzny, A. Zur Methodik der klinischen Blutdruckmessung. Centralbl. f. in. Med., 1901, vol. xxii, p. 599.

John, Max. Ueber den arteriellen Blutdruck der Phthisiker. Zeitschr. f. diät. u. phys. Therap., 1902, vol. v, p. 275.

Jourdin, Ch., et Fischer, G. De l'importance pronostique et thérapeutique de la pression artérielle. Rev. de Med., 1902, vol. xxii, p. 945.

Kapsammer, Geo. Blutdruckmessungen mit dem Gärtner'schen Tonometer. Wien. klin. Wochenschr., 1899, vol. xii, p. 1279.

Kaufmann, J., und de Bary, W. Ueber die Einwirkungen Priessnitz'scher Einwickelungen auf den Blutdruck bei croupöser Pneumonie und diffuser Nephritis. Berl. klin. Wochenschr., 1888, vol. xxv, p. 557.

Marfan, M. C. B. De l'abaissement de la tension artérielle dans la phthisie pulmonaire. La Semaine Med., 1891, vol. xi, p. 213.

Mercandino, F. Ueber den Einfluss einiger Herzstimulantien und einzelner hydriatischer Proceduren auf den Blutdruck im Verlaufe der akuten Infectionskrankheiten. Blätter f. klin. Hydrother., 1900, vol. x, p. 241.

Mosen, R. Ueber das Verhalten des Blutdrucks im Fieber. Deutsch. Arch. f. klin. Med., 1894, vol. lii, p. 600.

Naumann, H. Blutdruckmessungen an Lungenkranken. Zeitschr. f. Tuberkulöse, 1903, vol. v, p. 118.

Neisser. Ueber Blutdruckmessung am Krankenbett. Berl. klin. Wochenschr., 1900, vol. xxxvii, p. 557.

Norris, G. W. A Contribution to the Study of the Human Blood-pressure in some Pathological Conditions. Am. Jour. of the Med. Sci., 1903, vol. cxxv, p. 888.

Pässler, H. Experimentelle Untersuchungen über die allgemeine Therapie der Kreislaufstörung bei acuten Infectionskrankheiten. Deutsch. Arch. f. klin. Med., 1899, vol. lxiv, p. 715.

Potain, C. La pression artérielle de l'homme à l'état normal et pathologique. Paris, 1902.

Quirin, A. Ueber das Verhalten des normalen und pathologisch gesteigerten intraabdominalen Drucks und seine Rückwirkung auf die arterielle Blutcirculation. Deutsch. Arch. f. klin. Med., 1901, vol. lxxi, p. 79. (See pp. 99-102.)

Schüle. Ueber Blutdruckmessungen mit dem Tonometer von Gärtner. Berl. klin. Wochenschr., 1900, vol. xxxvii, p. 726.

Shaw, H. L. K. Arterial Tension in Childhood. Albany Med. Ann., 1900, vol. xxi, p. 88.

Thaussig, R. Zur Kenntniss der Gefässwirkung des Iods (bezw. Iodipin). Wien. med. Wochenschr., 1902, vol. lii, p. 1399.

Thayer, W. S. On the Late Effects of Typhoid Fever on the Heart and Vessels. Am. Jour. of the Med. Sci., 1904, vol. cxxvii, p. 391.

CHAPTER VIII

BLOOD-PRESSURE IN NERVOUS AND MENTAL DISEASES

1. Diseases of the spinal cord.

- A. Tabes dorsalis.

2. Diseases of the brain.

- A. Vascular diseases.
  - a. Cerebral hæmorrhage.
  - b. Cerebral thrombosis and embolism.
- B. Tumors and inflammations.
- C. General paresis.
- D. Myasthenia gravis.

3. The psychoses.

- A. Melancholia.
- B. Acute mania.
- C. Circular insanity.

4. Functional conditions.

- A. Epilepsy.
- B. Trigeminal neuralgia.
- C. Insomnia.
- D. Neuropathic individuals. Hysteria.

Literature.

CHAPTER VIII

BLOOD-PRESSURE IN NERVOUS AND MENTAL DISEASES

1. DISEASES OF THE SPINAL CORD

VERY little study has been made of the arterial tension in cord disease. All spastic conditions in which the arms are involved are, of course, excluded from observation by our clinical methods, on account of the influence of the heightened muscular tone on the transmission of the pressure (see page 61). In destructive lesions involving the higher segments, it would be of interest to know the extent to which the vaso-motor centres were damaged, so far as one might judge from the blood-pressure; but I know of no measurements under such conditions. Apart from this, it is hard to see what special significance the arterial pressure could have in this group of disease, except for the interesting facts which have come out in connection with locomotor ataxia.

A. Tabes Dorsalis.—Pal, in two recent articles, has thrown new light on vaso-motor manifestations in connection with the lightning pains and the visceral crises of tabetics. He examined the tension during the attacks by Gärtner's tonometer, and found that the lightning pains were attended by a constant fall in pressure, sometimes of considerable extent. In certain severe gastric and abdominal crises, however, an enormous rise occurred, up to 150 per cent. of the original pressure. One woman of twenty-eight, with 80 to 90 mm. between the paroxysms, had 170 to 190 mm. in moderate, 240 mm. in very severe attacks. A thirty-year-old man had 65 mm. pressure when free from pain, 140 mm. during the crisis. The climax of hypertension and of pain seemed to coincide, and both passed away together. Morphin caused sleep and a lessening of the pain, but no fall in pressure, and the heart often showed evidence of weakness after its use. Chloral, however, exerted a hypotensive, as well as an analgesic effect. Of special interest was the alternation of the abdominal and the lancinating pains. Whenever the latter came on, pressure promptly fell and the visceral crisis ceased.

On the strength of these findings Pal believes himself justified in assuming that a spasm of the splanchnic vessels is the cause of the suffering in a gastric crisis, and names this a vascular crisis (Gefässkrise). On the other hand, the irritation which brings on the lightning pains apparently affects depressor fibres in the posterior nerve roots, and this depressor stimulus is sufficient to interrupt or cut short an abdominal vascular crisis. This testimony is so important from the theoretical, as well as the practical therapeutic standpoint, that it is desirable that it should be corroborated or qualified by subsequent studies as speedily as possible.

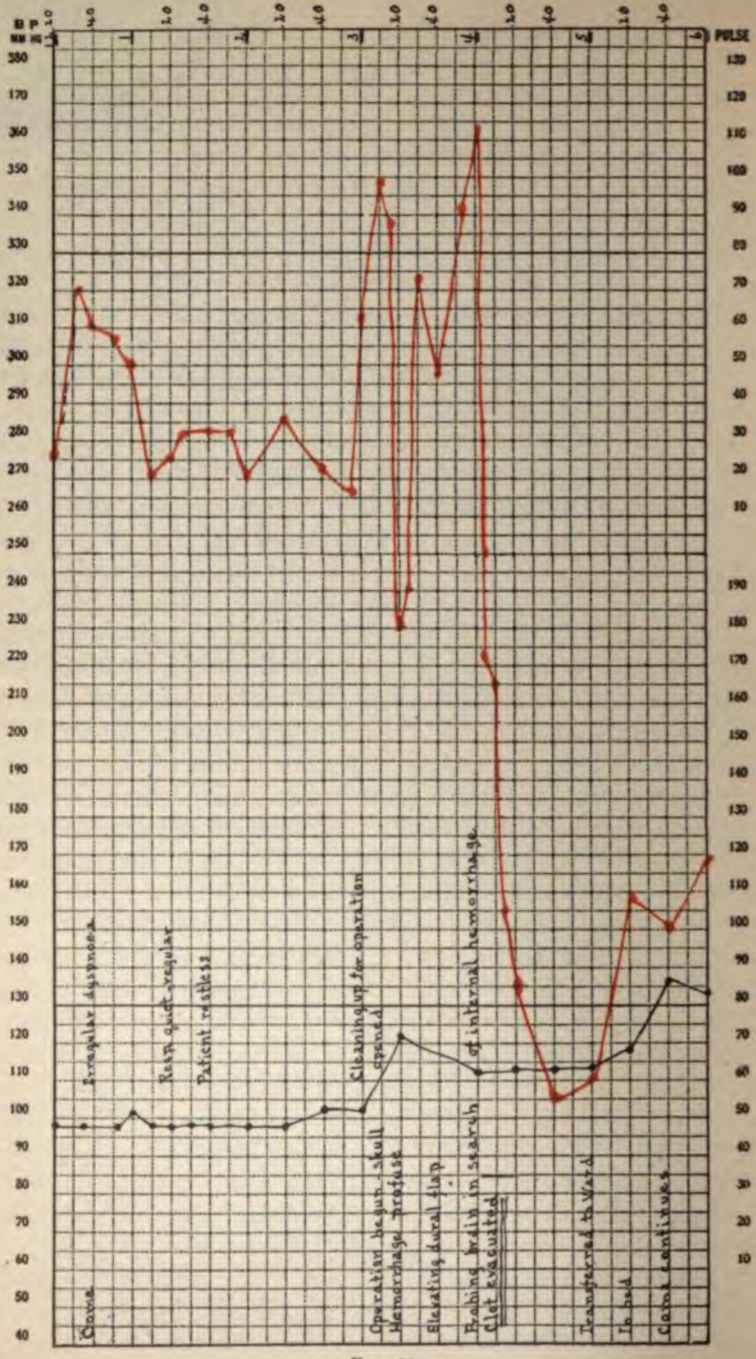
2. DISEASES OF THE BRAIN

A. Vascular Diseases.—The relation of cerebral arterio-sclerosis to general arterial disease has already been considered (see page 189). The sphygmomanometer gives no positive evidence of the existence of changes in the brain vessels, but is of distinct service where these are suspected, by revealing the presence or absence of essential hypertension, and suggesting its causative factors in kidneys, arteries, or heart. When hypertension is marked, cerebral hæmorrhage becomes more likely; when absent, thrombosis is the greater danger; so that in some degree both prognosis and treatment are facilitated by a knowledge of the arterial pressure. When, on the other hand, one of the vascular accidents has already occurred, the sphygmomanometer helps the differential diagnosis.

a. Cerebral Hæmorrhage.—Large hæmorrhages into the cerebral hemisphere are almost always attended by general compression symptoms, coma, slow pulse, and stertorous or Cheyne-Stokes breathing. Consequently they produce marked hypertension, which is more extreme the greater the rise in intracranial tension. The reasons for this have already been discussed (see page 140). It is of the greatest importance to know whether a comatose patient is in the initial or advanced stage of manifest cerebral compression, or has reached the period of terminal paralysis. No single bit of evidence tells this as clearly as the systolic blood-pressure. If this is extreme, above 300 mm. (5 cm.), 280 mm. (12 cm.), the medulla is undergoing serious compression, and danger to life is imminent. This is especially true if marked Traube-Hering waves can be measured, which is easily done by maintaining the pressure in the sphygmomanometer just above the level of systolic blood-pressure, and noting whether a succession of large pulse-waves come through periodically. With instruments giving diastolic pressure it is even simpler, for, with the pressure held at the diastolic level, the rhythmical variation in size of the pulse is striking. If, however, the symptoms are not urgent and the blood-pressure is not high, either the diagnosis of hæmorrhage is incorrect, or it is doing local rather than general harm. If the patient is evidently in serious condition, with deep coma, Cheyne-Stokes breathing, or cyanosis, and beginning respiratory failure, and a low or falling blood-pressure and rapid pulse are found, then the terminal stage has set in, and no treatment can much retard dissolution. Nothing could be more valuable in such cases than a blood-pressure record taken at frequent intervals, as Cushing, and Cook and Briggs have conclusively shown.

A high and rising blood-pressure indicates progressive failure of circulation in the medulla and an increasing hæmorrhage. On account of the circulus vitiosus established under such conditions, this rise in pressure is productive of more rapid bleeding; while an artificial reduction of tension by drugs, derivation, or bleeding, will be likely to kill the patient by the cessation of cerebral circulation, which results the moment the general arterial pressure falls below the intracranial. In consequence, some surgeons, especially Cushing in this country, advocate immediate operation, as for extradural hæmorrhage, with relief of intracranial tension and evacuation of the clot, if possible. In cases where the advance of the compression is studied by the sphygmomanometer, this certainly deserves trial. With a stationary or falling pressure, without increase in symptoms, it should not be thought of (see Fig. 62).

Hæmorrhage in the anterior fossa of the cranium will have the least effect on general blood-pressure, that in the posterior fossa most. Hill has shown how great pressure discontinuity



B P
MM HG
PULSE
10
40
1
20
40
2
20
40
3
20
40
4
20
40
5
20
40
6
380
170
360
350
340
330
320
310
300
290
280
270
260
250
240
230
220
210
200
190
180
170
160
150
140
130
120
110
100
90
80
70
60
50
40
130
120
110
100
90
80
70
60
50
40
30
20
10
190
180
170
160
150
140
130
120
110
100
90
80
70
60
50
40
30
20
10
Coma
Irregular dyspnoea
Resp. quiet irregular
Patient restless
Cleaning up for operation
Operation begun - skull opened
Hemorrhage profuse
Elevating dural flap
Probing brain in search of internal hemorrhage
Clot evacuated
Transferred to Ward
In bed
Coma continues

FIG. 62.

FIG. 62.—INTRACRANIAL HÆMORRHAGE (APOPLEXY) WITH EXPLORATION OF THE BRAIN AND EVACUATION OF A LARGE CLOT. (Cook's sphygm. 5 cm.)

Persistent extreme hypertension with slow (vagus) pulse, resulting from mechanically increased pressure on the medulla.

Exertion (preparation for operation) increasing the blood-pressure by exciting further hæmorrhage, and so increasing intracranial tension.

Elevation of bone flap, and brisk external hæmorrhage, reducing the blood-pressure momentarily.

Immediate complete relief of the hypertension on evacuation of the compressing intracerebral clot, with further depression due to slight shock, and subsequent return of blood-pressure to the normal level (arterio-sclerotic patient). (From Cook and Briggs, Chart No. XIX.)

exists between the cerebral and cerebellar chambers. It may be hoped that the sphygmomanometer will assist in the diagnosis of the rarer cerebellar hæmorrhages, which are sometimes puzzling. The most conspicuous posterior fossa hæmorrhage, in its influence on the medullary centres, is that accompanying fracture of the base of the skull. Extreme hypertension is a valuable diagnostic guide in this injury, sometimes overlooked, which occupies the border line between neurology and surgery.

Between uræmic and apoplectic coma the decision is more difficult, for a marked rise in pressure attends the former. Figures much over 300 mm. (R. R. 5 cm.) have seldom been reported, however, except in cerebral compression. A recent case of acute uræmia at City Hospital, in which no significant brain lesion was found at autopsy, but contracted kidneys with marked fatty change, had a systolic arterial pressure of 290 mm. (J. 12 cm.). Further data are necessary before definite statements can be made on this question. Here, as everywhere in medicine, the physician who gets in all the obtainable facts, including the blood-pressure, will less frequently go wrong than he who lays undue stress on any single diagnostic feature.

b. Cerebral Thrombosis and Embolism.—Obstruction of a brain artery can only affect blood-pressure directly, when the circulation of the medulla itself is suddenly interfered with. Such a lesion would be rapidly fatal. Indirectly, as Hill has shown, it may produce secondary compression through surrounding congestive or inflammatory œdema. Practical observations on arterial tension under such conditions are lacking. Moderate or considerable hypertension, from arterio-sclerosis or kidney disease, is not uncommon in the subjects of thrombotic hemiplegia (see Fig. 43), but I have never seen the high figures reached in hæmorrhage. Unquestionably the finding of approximately normal blood-pressure, early in a paralytic stroke, makes arterial occlusion highly probable. This is important for treatment. If hæmorrhage be excluded by the mode of onset, and extent of the local manifestations without general compression symptoms, any measures which lower general blood-pressure must be directly injurious, since they diminish the speed of the cerebral circulation and favor an extension of the clot. Far more are stimulant drugs, such as caffein, called for; and the effects should be controlled by systematic measurements of pressure.

B. Tumors and Inflammations.—Clinical observations of blood-pressure have not been reported in connection with growths or inflammatory brain lesions, except in operations for their removal. Hill thinks it probable that tumors cannot of themselves cause pressure symptoms, because room is made for their slow increase, as a rule, by atrophy of brain substance. Growths of the posterior fossa, especially when they obstruct the veins of Galen or the aqueduct of Sylvius, cause marked increase of intracranial tension and must be associated with high blood-pressure. The same should be true where considerable hæmorrhage into the growth takes place, or when there is much inflammatory œdema or exudation in tumors, meningitis, or abscess. Accurate information should be forthcoming.

C. General Paresis.—Pilcz has examined a large number of patients in every stage of general paralysis. He found normal tension in the earlier stages, 100 to 110 mm. (G.), falling as the disease advanced. In the terminal bedridden state hypotension became extreme, 50 to 80 mm. (G.). This seems analogous to the subnormal pressure of other wasting diseases, but is rather more pronounced. In several patients he was able to foretell death by the rapid fall in pressure. Where Bright's disease was present, hypertension was in evidence, and depressive mental states brought about a rise. The remissions in symptoms, so common in this disease, were accompanied by higher tonometer readings.

The only other report on general paresis is from Craig, who himself admits that his cases were not numerous or suitable enough to allow of conclusions.

D. Myasthenia Gravis.—Steinert reports a case of myasthenia gravis, in which the systolic pressure was low, 100 to 120 mm. (R. R. 5 cm.). The patient had a fall in pressure, with subsequent rise, in a cool Nauheim bath, which caused a well-marked elevation of tension in a healthy man. The observation is a purely isolated one. It is quoted here because the condition is described in neurological text-books, though its real pathology is not yet certain.

3. THE PSYCHOSES

Numerous interesting studies on blood-pressure in the insane have come from various asylums, here and abroad. The results are surprisingly uniform.

A. Melancholia.—The height of arterial pressure in melancholia seems to bear a definite relation to the intensity of mental suffering, and always tends to be above the normal average. Cramer gives some interesting case histories, with charts, which show this connection clearly (see Fig. 63). His work was with the v. Basch sphygmomanometer, but Alexander, and Craig, with the Hill and Barnard, Pilcz, and Rosse, with the tonometer, and Dunton, with the Riva-Rocci, all confirm it. Alexander distinguishes four types of melancholia: simple melancholia, in which no marked elevation of pressure occurs; an acute passive type, in which pressure is invariably high, decreasing as the symptoms abate, and rising with each relapse; an acute demonstrative type, with more irregular course, not so marked hypertension, nor so constant a descent with the amelioration of symptoms; lastly, chronic

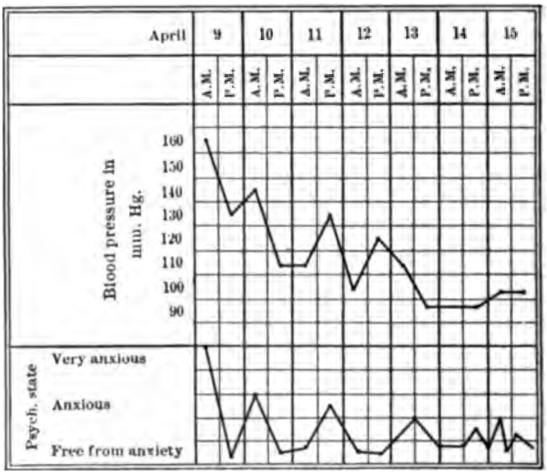


FIG. 63.—RELATION BETWEEN BLOOD-PRESSURE AND PSYCHICAL STATE IN MELANCHOLIA. (v. Basch's sphygm.)

Chart from a woman of forty, with severe melancholia of one year and one quarter duration, who improved during the period represented, becoming paranoiac later. (From Cramer, Curve I.)

melancholia, with low tension except during acute exacerbations of the mental symptoms. Craig says that tension is variable in agitated melancholia, corresponding with Alexander's demonstrative cases. Some patients in stupor he found had high readings. Pilcz saw readings as high as 130 to 200 mm. (G.) during paroxysms of anguish. As regards the causation of hypertension during intense mental depression, it seems reasonable to refer it to abnormal vaso-constrictor tone. Cramer sees in the high pressure the source of the acute mental suffering—a most unlikely theory, since hypertension has no such sequence in its most marked forms. Alexander believes it a manifestation of toxæmia from retained waste products. We certainly do not know enough of the morbid physiology of the psychoses to hazard an explanation of this rather constant feature. One thing it seems to indicate is, that acute melancholia should be considered an active mental state. Craig considers the sense of pressure on the top of the head, in melancholics, a vascular symptom. So far as treatment goes, Craig reports improvement in some patients from nitroglycerin or erythrol tetranitrate. He also noted a regular daily fall in pressure, with abatement of the anxiety, toward evening.

B. Acute Mania.—States of motor excitement have the opposite effect on blood-pressure from conditions of mental anxiety and motor depression. In mania the tension is regularly low. Pilcz found that persons with periodic mania had 25 to 30 mm. lower pressure during the excited state. In the exhaustion following a maniacal outbreak, Craig, and Alexander found hypotension more marked. The latter did not see subnormal readings in mania, but low as contrasted with melancholia. Dunton thinks in either case the motor condition has greater influence on the blood-pressure than the mental.

Baths of long duration, according to Rosse, are followed by a rise in pressure averaging 20 mm. (G.), and exert a quieting effect on the patient. Craig made a similar observation for baths of seven to eight hours' duration. When the bath was ineffective, the pressure remained unchanged.

C. Circular Insanity.—In circular insanity the findings for conditions of excitement and of depression hold good in the main. Pilcz saw a few exceptions to this rule, and found great variability of pressure at different periods, but considers the association of restlessness with low pressure usual. He thinks hypertension a factor in the differential diagnosis of stupor, occurring with it in circular insanity, but being absent in catatonic stupor.

4. FUNCTIONAL CONDITIONS

A. Epilepsy.—In epileptic, as in other convulsions, a marked rise in pressure is said to take place. One can scarcely see how accurate readings are made during the fit. Pilcz, in two cases, found a rapid fall as soon as muscular relaxation set it, reaching normal in a few minutes. Féré found much the same, though not so prompt a subsidence. His figures, using Bloch's instrument, are of little worth. The condition of the blood-pressure after a convulsion might be of considerable significance in distinguishing epilepsy from acute uræmia, in which hypertension persists during the coma and is a permanent feature.

B. Trigeminal Neuralgia.—Like most acute pain from other causes, the paroxysms of this agonizing tic cause a rise in blood-pressure. The height reached seems fairly proportional to the intensity of the suffering. There is no coincident change in pulse-rate, so that a pure reflex vaso-constriction seems the probable cause. One of Cushing's charts, from an operation for excision of the Gasserian ganglion, shows the effect of two paroxysms, before the administration of ether. The mild one produced a rise in systolic pressure from 160 mm. to 190 mm., the severe one, from 165 mm. to 235 mm. (R. R. 5 cm.).

C. Insomnia.—Bruce has made interesting studies of blood-pressure in sleepless patients, on the basis of which he distinguishes insomnia with high blood-pressure, 130 to 150 mm. (H. & B.), from insomnia with normal or low pressure. The first group always had a prompt fall in pressure when they went to sleep. Erythrol tetranitrate, in these patients, acted as a hypnotic if it reduced tension. This makes a relation between the hypertension and the insomnia manifest. Paraldehyde, in dose of ʒij, was the most certain and prompt drug in this group, sulphonal and trional were less trustworthy, and bromide had no effect. Sleep was possible with hypertension, but always produced some fall. For the cases with normal pressure, sulphonal, trional, and small doses of paraldehyde were most efficient. Kornfeld made comparative measurements on the same persons in natural sleep and that induced by trional. He uniformly observed a much greater fall after the drug, in some cases as much as 50 per cent. of the previous tension. When sleep did not follow, much less reduction of pressure occurred. After its continued use, a lowering of the average daily systolic blood-pressure was noted. Kornfeld thinks the hypotensive effect due to the quieting of the nervous system—that is, the removal of reflex vaso-constriction from psychical causes. This seems reasonable. It suggests the great value which sleep may have in cardiac cases, reducing the work of the heart, besides permitting repair of nervous tissues. In no diseased condition, I think, is the beneficial effect of a night's rest from hypnotics so clearly visible.

D. Neuropathic Individuals. Hysteria.—Heim examined 25 normal and 58 neuropathic children, finding considerably higher tonometer readings in the latter, 130 to 150 mm. He thinks it valuable in the frequently difficult task of deciding on the reality of children's complaints, higher pressure indicating greater psychical irritability and suggesting a neurotic basis. He observed no difference between neurasthenia and hysteria.

Rosse found that hysterical girls, who complained of pain and other abnormal sensations during menstruation, had a steady rise of 20 mm. during the flow, instead of normal or diminished pressure. Between the monthly periods the pressure was not only lower, but fluctuated rapidly. An increase in pulse-rate attended the menstrual rise in pressure. Pilcz notes a cataleptic boy, bedridden and profoundly emaciated, who preserved normal blood-pressure. I have seen markedly neurasthenic persons with rather low normal systolic pressure, and diminished pulse-pressure. I am inclined to doubt the constancy of high tension in the neurotic. One must be especially careful not to confuse the neurasthenia which is sometimes due to vascular or kidney disease, with its essential hypertension, and these reported high normal readings in simple neurasthenics.

LITERATURE

Alexander, H. de M. A Few Observations on the Blood-pressure in Mental Disease. Lancet, 1902, vol. ii, p. 18.

Bruce, L. Some Observations upon the General Blood-pressure in Sleeplessness and Sleep. Scottish Med. and Surg. Jour., 1900, vol. vii, p. 109.

Cook, H. W., and Briggs, J. B. Clinical Observations on Blood-pressure. Johns Hopkins Hosp. Rep., 1903, vol. xi, p. 451.

Craig, M. Blood-pressure in the Insane. Lancet, 1898, vol. i, p. 1742.

Cramer, A. Ueber Verhalten des Blutdrucks während der Angst der Melancholischen. Münch. med. Wochenschr., 1892, pp. 82 and 110.

Cushing, Harvey. The Blood-pressure Reaction of Acute Cerebral Compression, Illustrated by Cases of Intracranial Hæmorrhage. Am. Jour. of the Med. Sci., 1903, vol. cxxv, p. 1017.

—— On the Avoidance of Shock in Major Amputations by Cocainization of Large Nerve-Trunks Preliminary to their Division. With Observations on Blood-pressure Changes in Surgical Cases. Ann. of Surgery, 1902, vol. xxxvi, p. 337, Fig. 12.

Dunton, W. R., Jr. Some Observations upon Blood-pressure in the Insane. Boston Med. and Surg. Jour., 1903, vol. cxlix, p. 422.

Féré, Ch. La pression artérielle dans les paroxysmes epileptiques et dans le colère. Compt. rend. de la Soc. de Biol., 1889, vol. i, p. 368.

Heim, P. Das Verhalten des Blutdrucks bei neuropathischen Kindern. Deutsch. med. Wochenschr., 1900, vol. xxvi, p. 321.

Hill, Leonard. The Physiology and Pathology of the Cerebral Circulation. London, 1896, pp. 182–200.

Kornfeld, S. Erfahrungen über Trional als Schlafmittel, mit besonderer Rücksicht auf die Beeinflüssung des Blutdrucks. Wien. med. Blätter, 1898, vol. xxi, pp. 3, 25 and 40.

Pal, J. Ueber Gefässkrisen und deren Beziehung zu den Magen und Bauchkrisen bei Tabiker. Münch. med. Wochenschr., 1903, vol. L², p. 2135.

—— Die vasomotorischen Begleiterscheinungen der lanzinierenden Schmerzen und das Alternieren tabischer Krisen. Wien. med. Wochenschr., 1904, vol. liv, p. 2.

Pilcz, A. Ueber einige Ergebnisse von Blutdruckmessungen bei Geisteskranken. Wien. klin. Wochenschr., 1900, vol. xiii, p. 276.

Rosse. Beitrag zur Blutdruckmessung bei Geisteskranken. Allgem. Zeitschr. f. Psychiat., 1903, vol. lix, p. 737.

Steinert, H. Ueber Myasthenie und myasthenische Reaktion. Deutsch. Arch. f. klin. Med., 1903, vol. lxxviii, p. 346.

CHAPTER IX

BLOOD-PRESSURE IN SURGICAL CONDITIONS

1. Uses of the sphygmomanometer in surgical practice.

2. Blood-pressure during surgical operations.

- A. Influence of the anæsthetic.
  - a. Ether.
  - b. Chloroform.
  - c. Nitrous oxide.
  - d. Cocain.
- B. Influence of the operative procedures.
  - a. Peripheral operations.
  - b. Operations on the abdominal cavity.
  - c. Gynæcological operations.
  - d. Operations on the thoracic cavity.
  - e. Operations on the cranial and vertebral cavities.
  - f. Operations on the genito-urinary organs.

3. Blood-pressure in surgical accidents and diseases.

- A. Hæmorrhage.
  - a. Treatment.
- B. Collapse and shock.
  - a. Prophylaxis.
  - b. Treatment.
- C. Head injuries.
- D. Acute peritonitis
- Literature.

CHAPTER IX

BLOOD-PRESSURE IN SURGICAL CONDITIONS

1. USES OF THE SPHYGMOMANOMETER IN SURGICAL PRACTICE

THE evident practical value of blood-pressure records during operation, and in the study of certain surgical problems, has been so ably presented by Crile, and Cushing, that the sphygmomanometer is finding almost as wide acceptance among surgeons as among medical men. The illuminating experiments of Crile (see page 157) upon shock, and of Cushing on states of increased intracranial tension (see page 139), make its use imperative for the intelligent treatment of these two conditions. As a guide to the anæsthetist, the arterial pressure seems more valuable than the pulse alone. Both together are of the utmost prognostic significance. Beyond these direct results, I am inclined to believe that pre-operative observations of blood-pressure would afford indications of cardio-vascular disease, in some patients, which might well influence the decision as to the wisdom of operative interference. Such a use, I fear, might not be a universal recommendation.

2. BLOOD-PRESSURE DURING SURGICAL OPERATIONS

Systematic measurement of the blood-pressure during operation is being widely adopted by surgeons, and bids fair to become general. It may easily be carried out by the anæsthetist. To be of value, a determination should be made every five minutes, and, with the pulse-rate, recorded on a chart which can be seen by the operator. This procedure was originally recommended by Cushing. The chart described heretofore is well adapted for this purpose, and is more easily read if the systolic pressure be marked with a red pencil, the pulse in blue or black. The condition of the patient's circulation can then be taken in at a glance. Systolic pressure alone is necessary, because the object is merely to follow the changes in tension. It can be measured more quickly than diastolic, and is more affected by hæmorrhage and other causes of hypotension. In order to utilize such a graphic record, the surgeon must know what the ordinary influence of the various steps in a surgical operation are, and what the extraordinary and dangerous manifestations. These will be taken up in order, so far as possible.

A. Influence of the Anæsthetic. a. Ether.—All observers agree that ether, even in large amounts, seldom produces a significant fall in blood-pressure. This agrees with the evidence in animals, very large quantities of ether being required to seriously affect heart or vaso-motor system. Duplay and Hallion have recently shown this anew, and called attention to the blood-pressure as the one sure danger signal. In the first and second stages the pressure usually rises, due to the excitement and muscular exertion, but in full surgical anæsthesia it falls to near the normal. According to Cook and Briggs, it never produces marked hypotension unless causes for shock, or cardiac failure, are present. As the patient comes out of narcosis, the pressure curve rises. Blauel tabulates 100 administrations, watched with the tonometer. In 79 per cent. there was a rise in pressure during the whole period; in 9 per cent. no change or an insignificant fall. Only in 12 per cent. of the cases was there a real fall, and these were mainly weak persons, or with severe hæmorrhage or profuse sweating (see Figs. 64, 67, and 69).

b. Chloroform.—The danger of sudden death during chloroform narcosis has always been emphasized in this country, though often denied abroad. Blood-pressure observations certainly put the fear of chloroform on a sound footing. Except during labor (Cook and Briggs), it is usually attended by diminished arterial tension from the start. Duplay and Hallion, and Remedi, obtained the same result in animals. Blauel, in 39 cases, found a marked fall in 69 per cent., a moderate fall in 18.9 per cent. Only 10.8 per cent. showed a continued rise in pressure throughout narcosis, and 8.1 per cent. an average rise. Kapsammer gives very similar figures for 80 cases, only

5 showing a real continued rise. The diminution in tension was usually 10 to 40 mm. (G.); a few times 60 to 70 mm.; once 120 mm. Puricelli saw some increase in tension during the preparation for operation, which continued during the first stage of anæsthesia, after which it regularly decreased. Neither he nor Kapsammer could discover any relation between the previous height of blood-pressure and the tolerance of the anæsthetic. Awaking from anæsthesia, coughing, or vomiting increased the pressure by 20 to 30 mm. Blauel's chart (Fig. 64), which shows an average curve for both ether and chloroform, computed from a number of cases, is more instructive than any single observation (see Fig. 70).

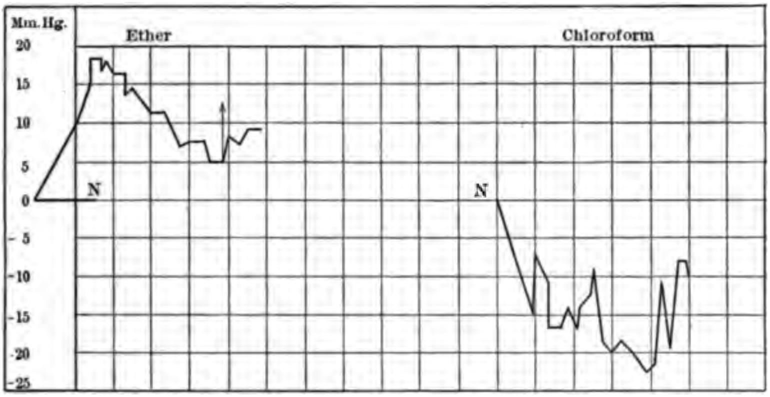


FIG. 64.—AVERAGE CURVES OF BLOOD-PRESSURE DURING ANÆSTHESIA.
(Gärtner's tonometer 1.5 cm.)

Left, for ether, calculated from 25, and right, for chloroform, calculated from 18 individual curves of healthy persons over twenty years old, during narcosis of at least fifty minutes. The horizontal N signifies the normal height of pressure. (From Blauel, Fig. 6.)

c. Nitrous Oxide.—Laughing gas, as one might expect, causes some hypertension, from the partial asphyxia it induces. According to Cook and Briggs, when given with ether it eliminates the hypertension of the second stage of excitement, the patient passing directly from the primary rise to the lower pressures of deep narcosis. This is evident in several of the accompanying charts (see Figs. 68 and 71).

d. Cocain.—Crile reports that abdominal operations under local anæsthesia produce less depression of blood-pressure than those done with a general anæsthetic. Of course protracted and extensive operations were impossible, and the slight effect on blood-pressure was probably due in large part to the small amount of manipulation involved. When pain or fear was produced, tension rose irregularly, with subsequent decline (see Fig. 65). Cook and Briggs found that cocain, administered hypodermically, usually produced a distinct rise in pressure during collapse and shock, and seemed a fairly reliable vaso-motor stimulant. Infiltration anæsthesia must also increase blood-pressure temporarily, when considerable tension of the tissues is developed. The cocainization of large nerve-trunks will be considered under the prophylaxis of shock.

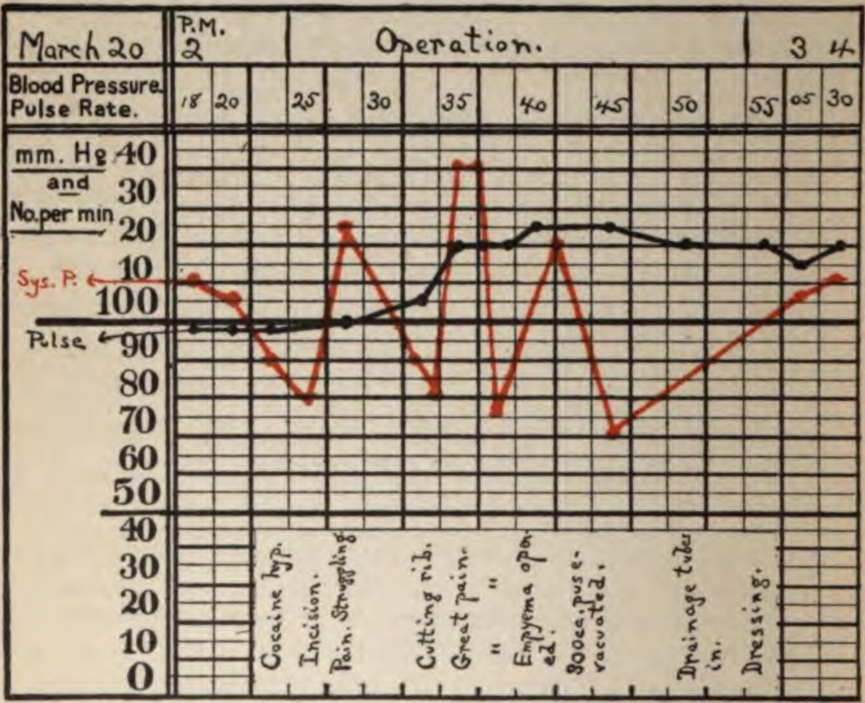


FIG. 65.—THORACOTOMY FOR POST-PNEUMONIC EMPYEMA UNDER LOCAL COCAIN ANÆSTHESIA. (Author's sphygm. 12 cm.)

Note the great rise in pressure with each cutting act, especially when accompanied by pain (reflex vaso-constriction).

Operation by Dr. Howard Collins, City Hospital. Observation by Dr. A. H. Garvin.

Cocainization of the spinal cord, according to Cushing, may induce dangerous hypotension, through paralyzing, in the upper dorsal region, the efferent nerve-fibres which control the splanchnic circulation. Application of cocain to the medulla oblongata, in Crile's experiments, reduced the blood-pressure to 40 or 50 mm., and rendered the centre inactive to reflex stimulation. Apart from the possible direct vaso-motor paralysis, these operations show little pressure variation (see Fig. 66).

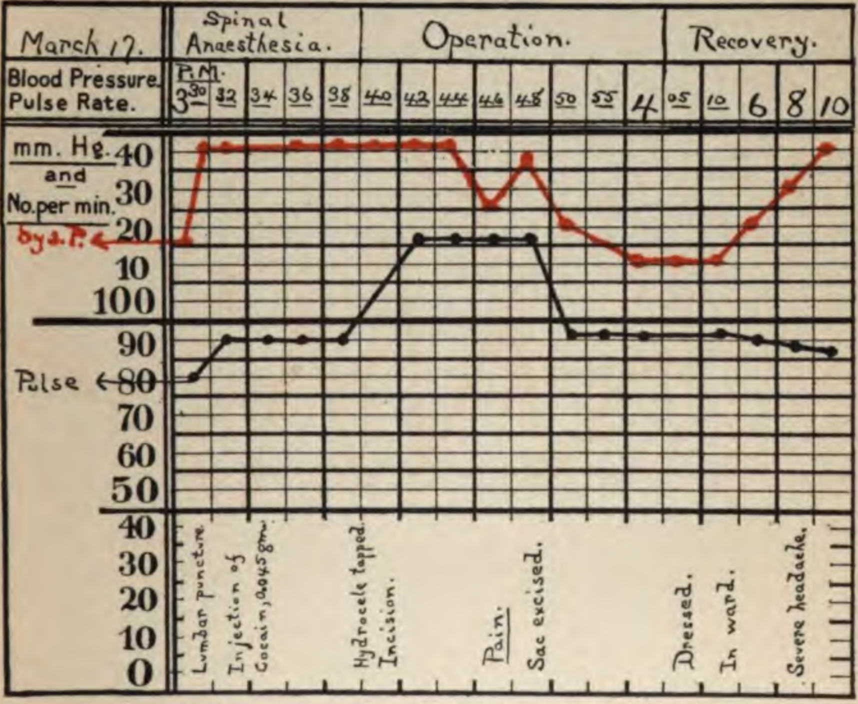


FIG. 66.—EXCISION OF HYDROCELE SAC AFTER SPINAL COCAINIZATION.
(Author's sphygm. 12 cm.)

Note the absence of any special change in pressure during the operation, after the primary rise, due to the pain or excitement.

Operation by Dr. Eugene Fuller, City Hospital. Observation by Drs. A. H. Garvin and Garside.

B. Influence of the Operative Procedures. a. Peripheral Operations.—All cutting involves the irritation of peripheral nerve-endings, which, as a rule, provokes reflex vaso-constriction, just as in the laboratory animal. The slight rise in the blood-pressure curve, due to the primary incision, is visible on all charts of operations. It seldom amounts to more than 10 mm. In shock alone this pressor effect of peripheral irritations is absent, and blood-pressure is unchanged, or shows reflex depression.

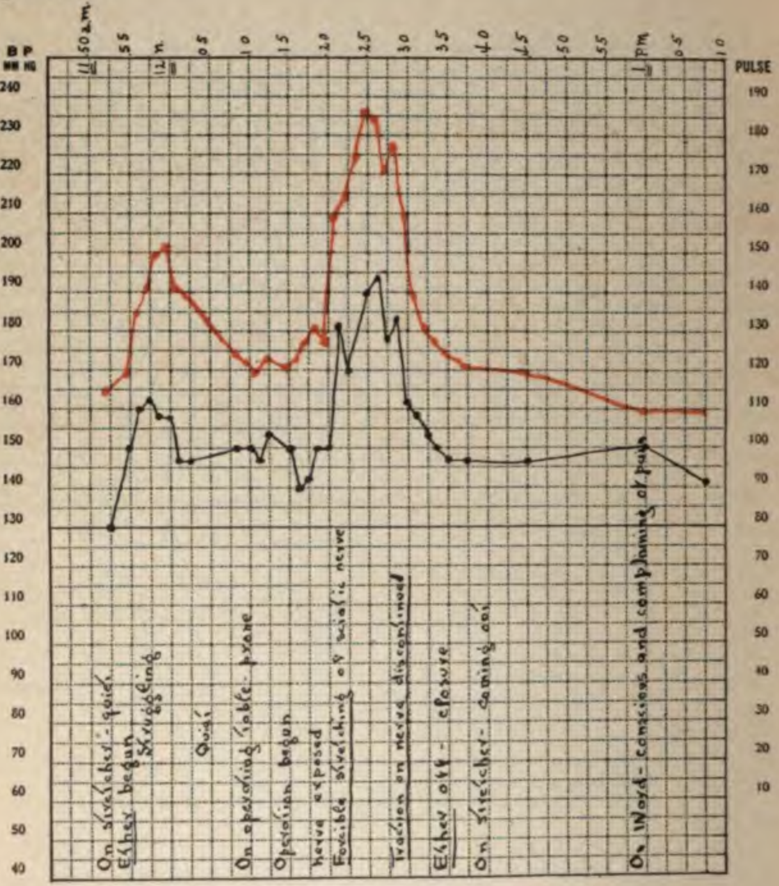


Fig. 67.—Forcible stretching of left sciatic nerve for sciatica. (Cook's sphygm. 5 cm.)

Primary "ether rise" in blood-pressure, with slight acceleration of the pulse-rate. Marked rise in blood-pressure, with greater acceleration of the pulse-rate, during the manipulation of the nerve (transient pressor effect of peripheral nerve stimulation). (From Cook and Briggs, Chart No. II.)

A much greater reflex rise in arterial pressure follows the irritation of nerve-trunks. This is typically shown in Fig. 67, from stretching of the sciatic nerve. It is a clinical duplicate of the tracing in Fig. 5, obtained on stimulation of the anterior crural nerve in a dog. A similarly marked vaso-constriction follows the simple operation of dilating the sphincter ani. Cushing suggests that the sudden deaths, which have occasionally been reported during these slight operations, may be due to rupture of a diseased cerebral vessel by the sudden hypertension.

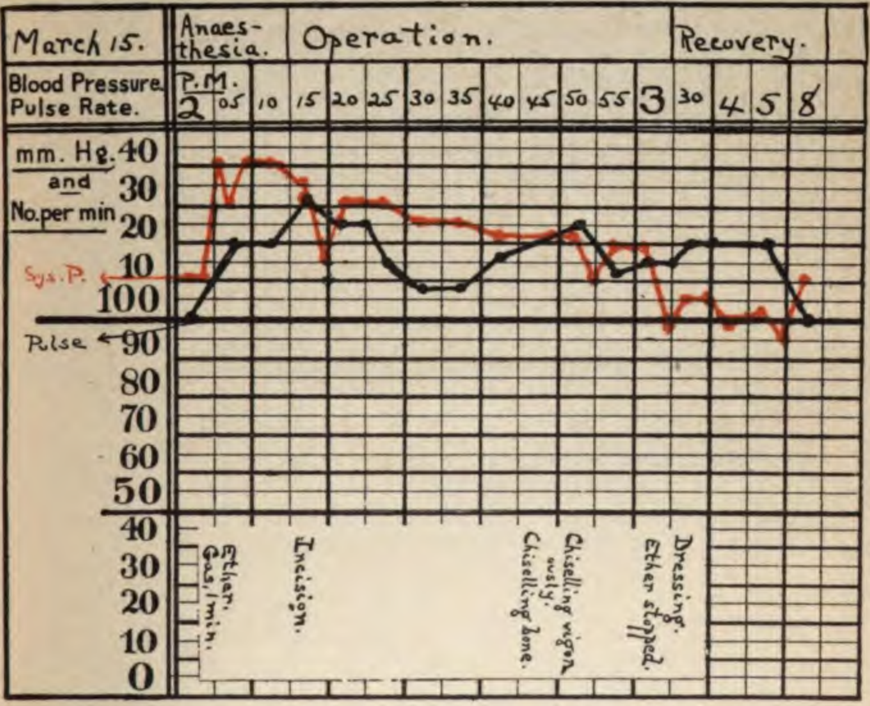


FIG. 68.—OPERATION FOR OSTEOMYELITIS OF FEMUR. (Author's sphygm. 12 cm.)

Note the primary rise in blood-pressure and pulse-rate from nitrous oxide; the absence of any further rise from ether; the fall, as full anæsthesia was reached; the incision rise (15 mm.); the slight subsequent effect of the operative procedures.
Operation by Dr. Howard Collins. Observation by Dr. Chapin.

Severe manipulation of large mixed or sensory nerves, as in forced retraction, the separation of firmly fixed tumors, and other procedures requiring considerable traction, may cause a sharp fall in blood-pressure. In Crile's experience this was especially pronounced when the superior laryngeal nerve was injured. A reflex slowing of the heart coincided. The same effect was produced by irritation of the laryngeal mucous membrane in animals, and operations on the larynx are frequently accompanied by dangerous shock. Kapsammer reports a fall from 110 mm. to 40 mm. (G.), with profuse sweating, due to traction on the sciatic nerve during an operation for osteomyelitis of the femur. Section of large nerve-trunks also has a serious depressor effect, and may lead to grave hypotension. Cushing, and Crile, both report such an occurrence in operations involving division of the brachial plexus.

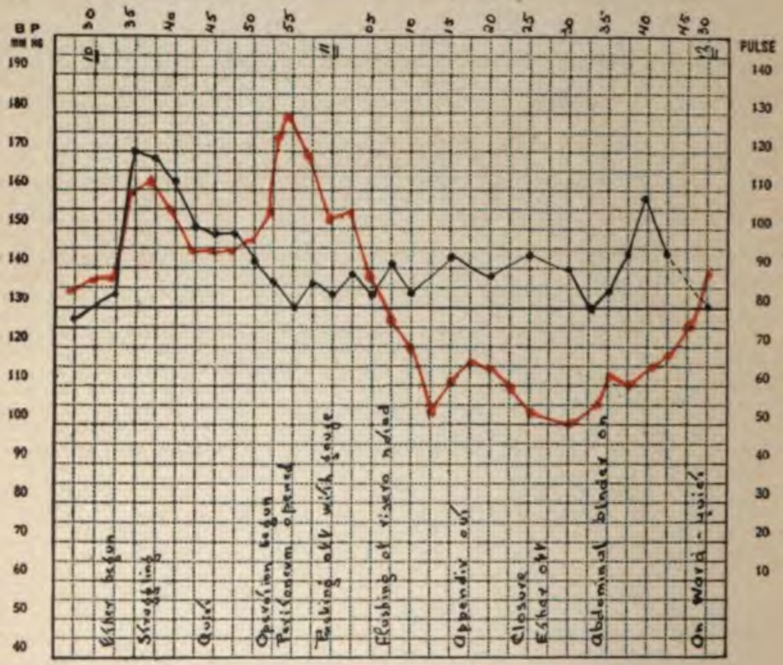


FIG. 69.—LAPAROTOMY (APPENDECTOMY). (Cook's sphygm. 5 cm.)

Initial etherization rise in blood-pressure.
Primary rise in blood-pressure from peritoneal irritation.
Secondary fall in blood-pressure from continued peritoneal irritation, coincident with splanchnic engorgement, and without change in pulse-rate. (From Cook and Briggs, Chart No. III.)

According to Crile, little variation of blood-pressure attends operations upon bone. Fig. 68 shows such a case. In the more extensive external operations, such as the complete Halstead operation for cancer of the breast, Cushing found that

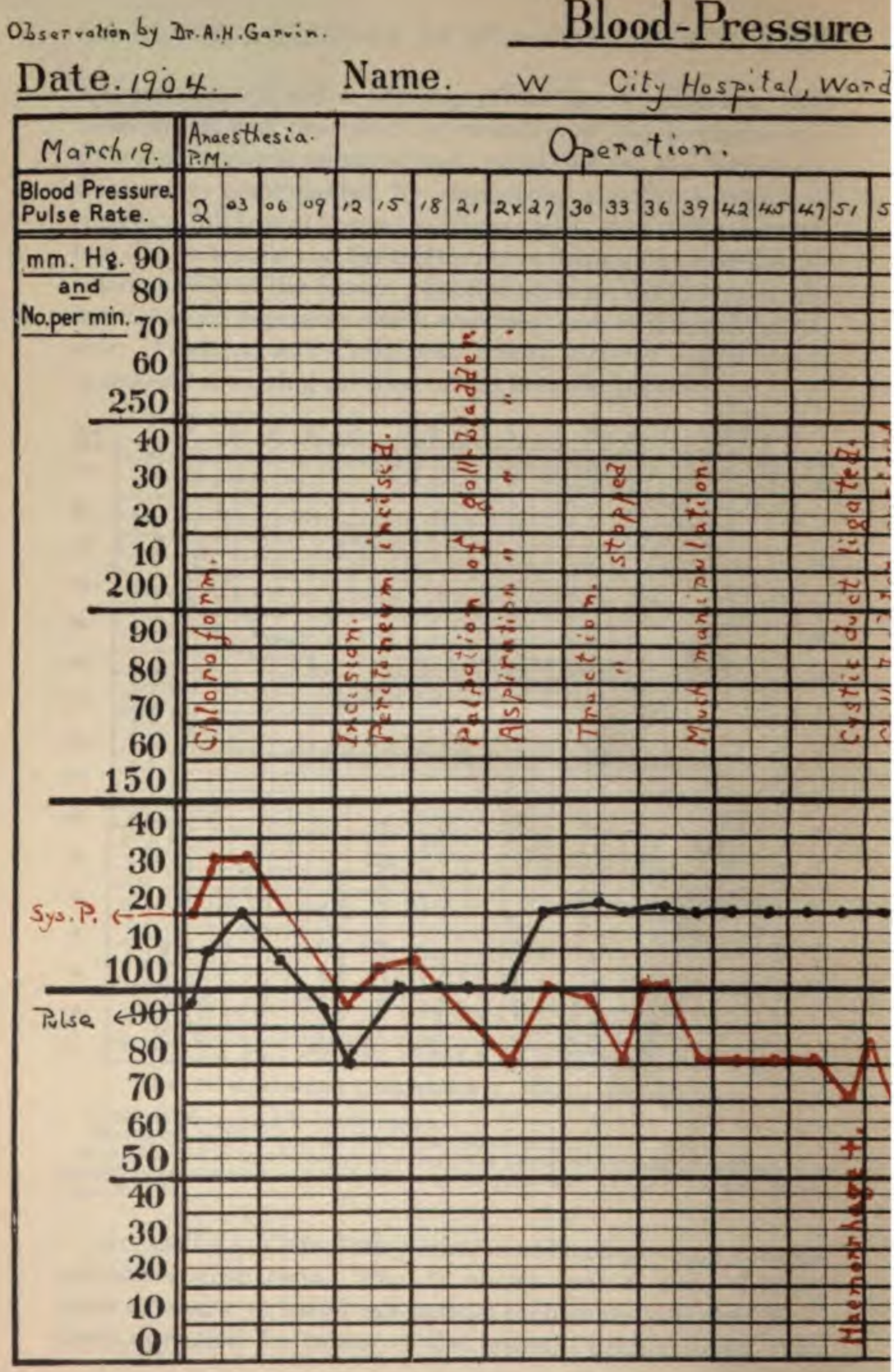
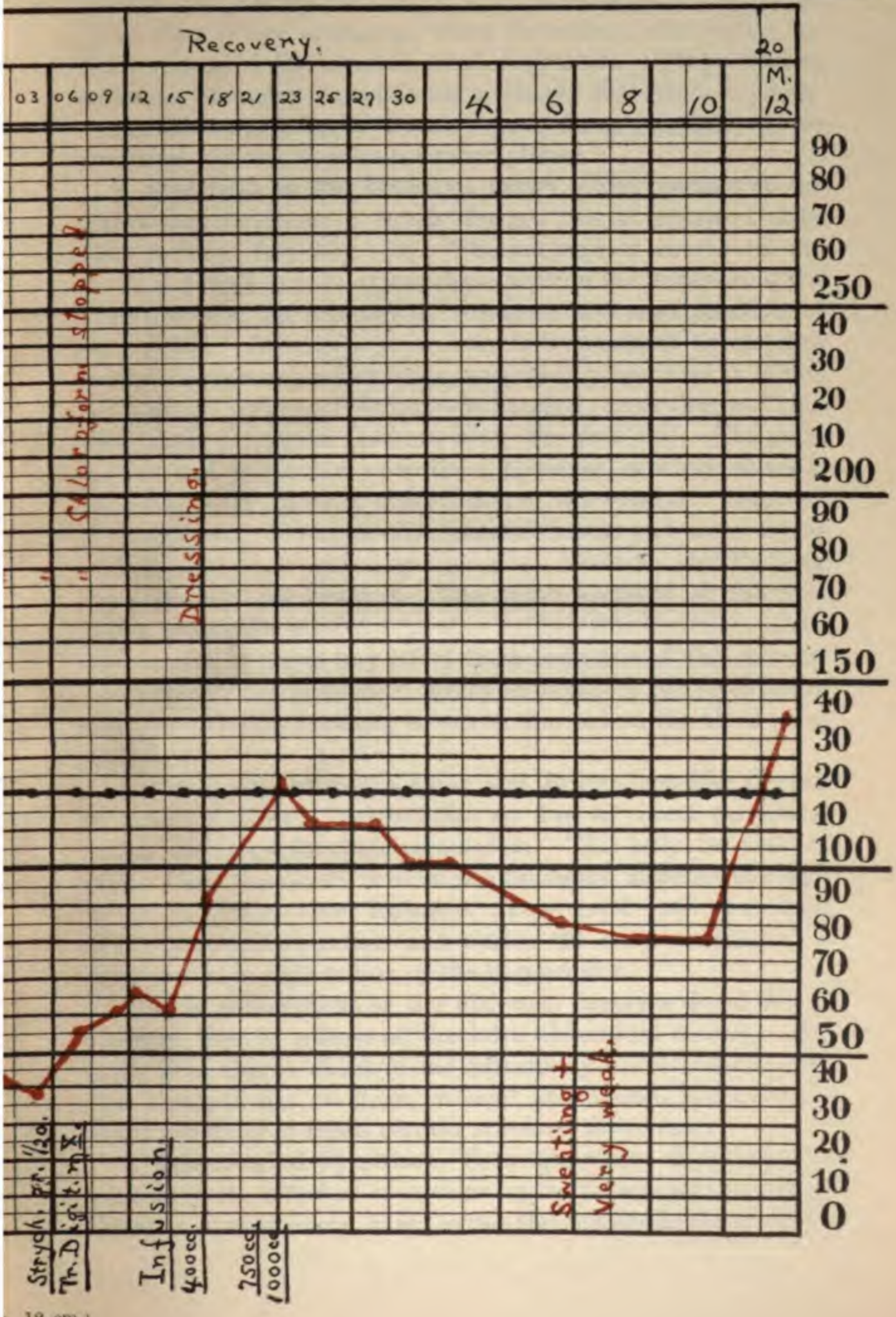


Fig. 70. (Author's

Pulse Chart.

Age. 44

Cholecystectomy by
Dr. Howard Collins.



. 12 cm.)

little fall in pressure resulted if hæmostasis was perfect; Crile found that rough sponging, blunt dissection, and strong retraction caused the most marked depression. These clinical instances of reflex vaso-dilatation should find their explanation in the knowledge of the depressor fibres in mixed nerves (see page 21), which is as yet incomplete.

b. Operations on the Abdominal Cavity.—The opening of the peritoneum produces a much sharper rise of pressure than skin incision (see Fig. 69). The subsequent course of the curve is downward, in extent dependent on the duration of the operation and the amount of exposure and manipulation of the viscera. Increased pulse-rate is a constant accompaniment. Simple appendectomies, and other procedures in which but a small portion of intestine is handled, according to Crile, show little change in pressure after the first fall. The more serious and protracted operations, however, in which shock is common, need constant observation of the tension. Removal of gangrenous bowel, in strangulated hernia and other forms of intestinal obstruction, sponging the intestine, and flushing the abdomen, all lower it. The upper segment of abdomen seems especially sensitive in this respect (see Fig. 70). Crile was unable to prove any effect from irritation of the mucous membrane of the intestine. Every irritation of the peritoneum after the primary incision, however, showed on the blood-pressure chart.

Cushing, and after him Cook and Briggs, consider the fall in pressure during laparotomies as due to direct peripheral action on the splanchnic circulation. This may be true in part. Crile, however, noted a simultaneous increase in respiration in almost every instance. This could only have been due to a medullary reflex, and makes the same mechanism a more probable explanation of the hypotension.

Simple paracentesis of the abdomen causes a slight fall in pressure, due to release of the intra-abdominal tension. Remedi, who thinks shock is due principally to chloroform, says that laparotomies on dogs, without anæsthesia, cause only a slight lowering of mean carotid pressure, 2 to 8 mm.

c. Gynæcological Operations.—Some difference of opinion obtains as to the effect of operations on the female pelvic organs. Crile, in almost every case, found the manipulation of these

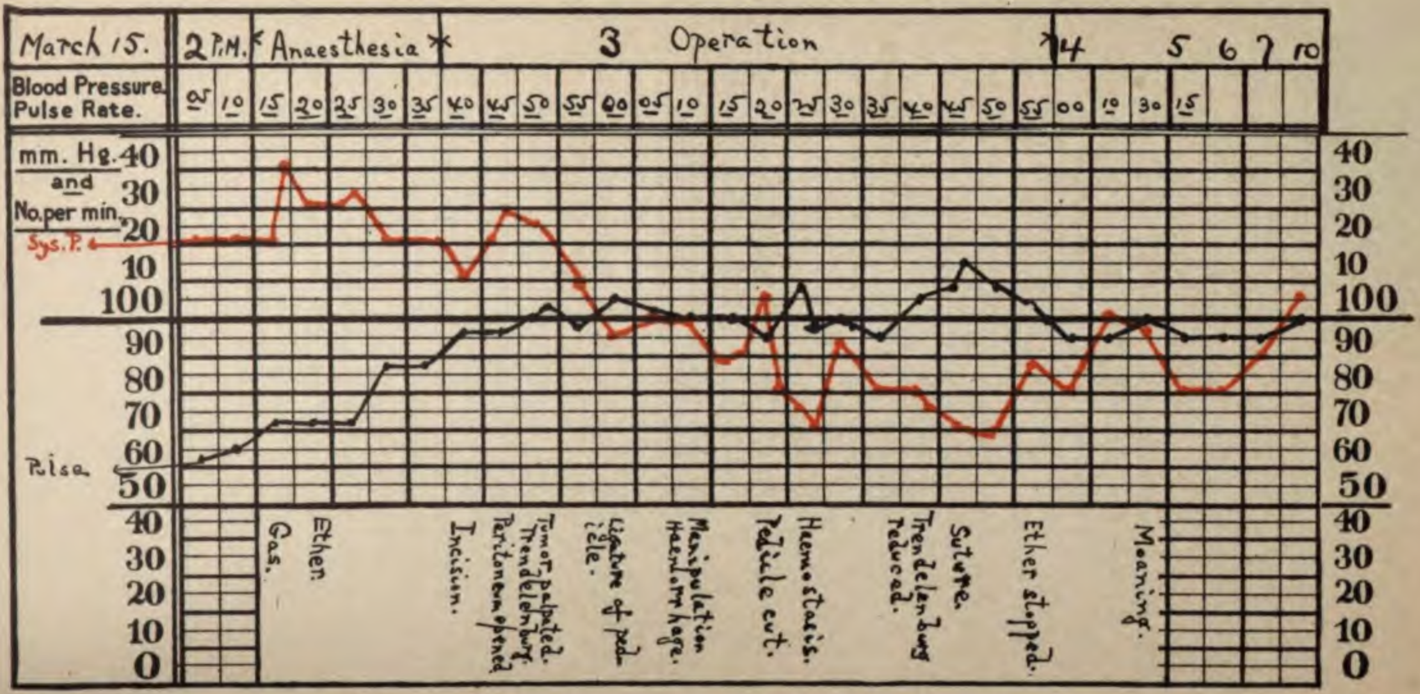


Fig. 71.—Laparotomy for uterine fibroids. (Author's sphygm. 12 cm.)

Note the rise in pressure during early anæsthetization, and from incision of skin and peritonæum; the marked fall produced by manipulation and hæmorrhage, with slow recovery.
Operation by Dr. H. S. Stearns. Observation by Dr. A. H. Garvin.

organs, whether from their vaginal or peritoneal aspect, produced a rise. The height was proportional to the severity of the traumatism. Schroeder, on the other hand, thinks these operations show a considerable lowering of pressure, those by the vaginal route much less than the abdominal. He uses chloroform narcosis, but thinks the laparotomy much more provocative of hypotension than the anæsthetic. Possibly the combined effect explains the variation of his results from Crile's. He advocates the systematic charting of blood-pressure, during and after operations, and considers a secondary fall, after the initial recovery of pressure succeeding operation, of serious prognostic significance. Fig. 71 illustrates an abdominal operation for uterine fibroids, in which considerable bleeding occurred.

d. Operations on the Thoracic Cavity.—Surgeons, like physicians, report a considerable lowering of tension on the removal of pleural exudates (Crile, Kapsammer). This shows in a small way in Fig. 65. Otherwise I know of no observations.

e. Operations on the Cranial and Vertebral Cavities.—The removal of bone, in trepanation or laminectomy, seems to exert little influence on blood-pressure. Incision of the dura with a sharp knife has no effect, but irritation of either cerebral or spinal dura mater causes a fall in pressure, most marked from sponging (Crile). The influence of the further steps depends on the underlying lesion. If there is no increased intracranial tension, incision of the cortex, or probing, are unaccompanied by marked change. If cerebral pressure is much increased by the operation, the typical blood-pressure reaction follows. Cushing shows this especially in his charts from operations on the Gasserian ganglion. Elevation of the temporal lobe, to expose the ganglion, produced a sharp rise in the curve. He reports one case, where fatal shock followed excision of the ganglion, with rapid fall in pressure.

When operation is undertaken for the relief of acute cerebral compression, whether from middle meningeal or intracerebral hæmorrhage, or fracture, an enormous drop in arterial pressure follows the release of the intracranial tension. Fig. 62, taken by Briggs from one of Cushing's cases, is typical of this. No other change in blood-pressure met with clinically is so striking. Hill, Kocher, and Cushing have all called attention to the necessity for extensive osteoplastic reaction of the skull in the treatment of general cerebral compression. A small opening is immediately occluded by the portion of cortex which bulges into it, and no good is done. In the terminal stage of compression, a falling blood-pressure may be arrested by immediate operation, even when artificial respiration has to be resorted to on the table, as Cushing has demonstrated.

In operations for tumor of the brain, Frazier emphasizes the importance of sphygmomanometric observation. He reports instances of surgical shock with typical hypotension, which developed during manipulation of the brain in such cases.

f. Operations on the Genito-urinary Organs.—Crile observed a fall in pressure from traction on the spermatic cord during herniotomy and operations on the testicle. Amputation of the penis in an elderly man gave a distinct fall. The removal of a large carcinomatous kidney was attended by marked hypotension. During all procedures in this field, depressor effects were manifested, if the blood-pressure was influenced at all. Fig. 72 shows an extreme fall in pressure during prostatectomy, in a patient with chronic interstitial nephritis. The considerable hæmorrhage acted like therapeutic venesection in relieving hypertension.

3. BLOOD-PRESSURE IN SURGICAL ACCIDENTS AND DISEASES

A. Hæmorrhage.—Loss of any considerable volume of blood, in an anæsthetized patient, results in an immediate descent of the blood-pressure curve, fairly proportional to the severity of the hæmorrhage. Unless extreme, or succeeded by shock, a compensatory rise follows. The predisposition to shock, which accompanies hypotension from acute anæmia, is its dangerous feature in most cases. Figs. 70, 71, and 72 illustrate the effects of bleeding as clearly as Fig. 6, taken in the laboratory.

a. Treatment.—The study of therapeutic measures for the relief of low blood-pressure after hæmorrhage must never neglect the definite physiological tendency of the blood-pressure to return unaided to its normal level. No interference can be accorded any value unless it obviously hastens this return. Saline infusion, either intravenous or subcutaneous, seems to

Observation by Dr. A. H. Garvin.

Blood-Pressure

Date. 1904. Name. D. City Hospital. W

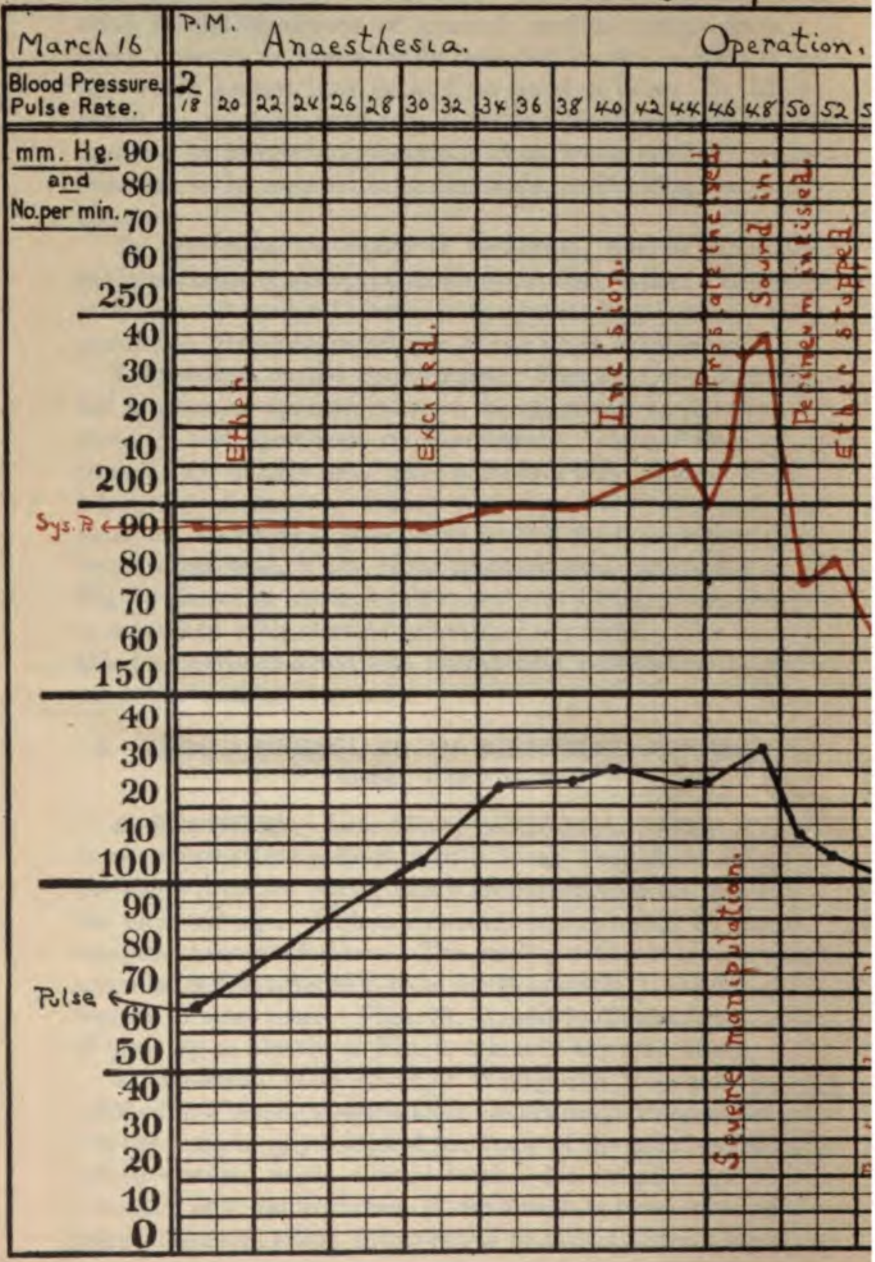
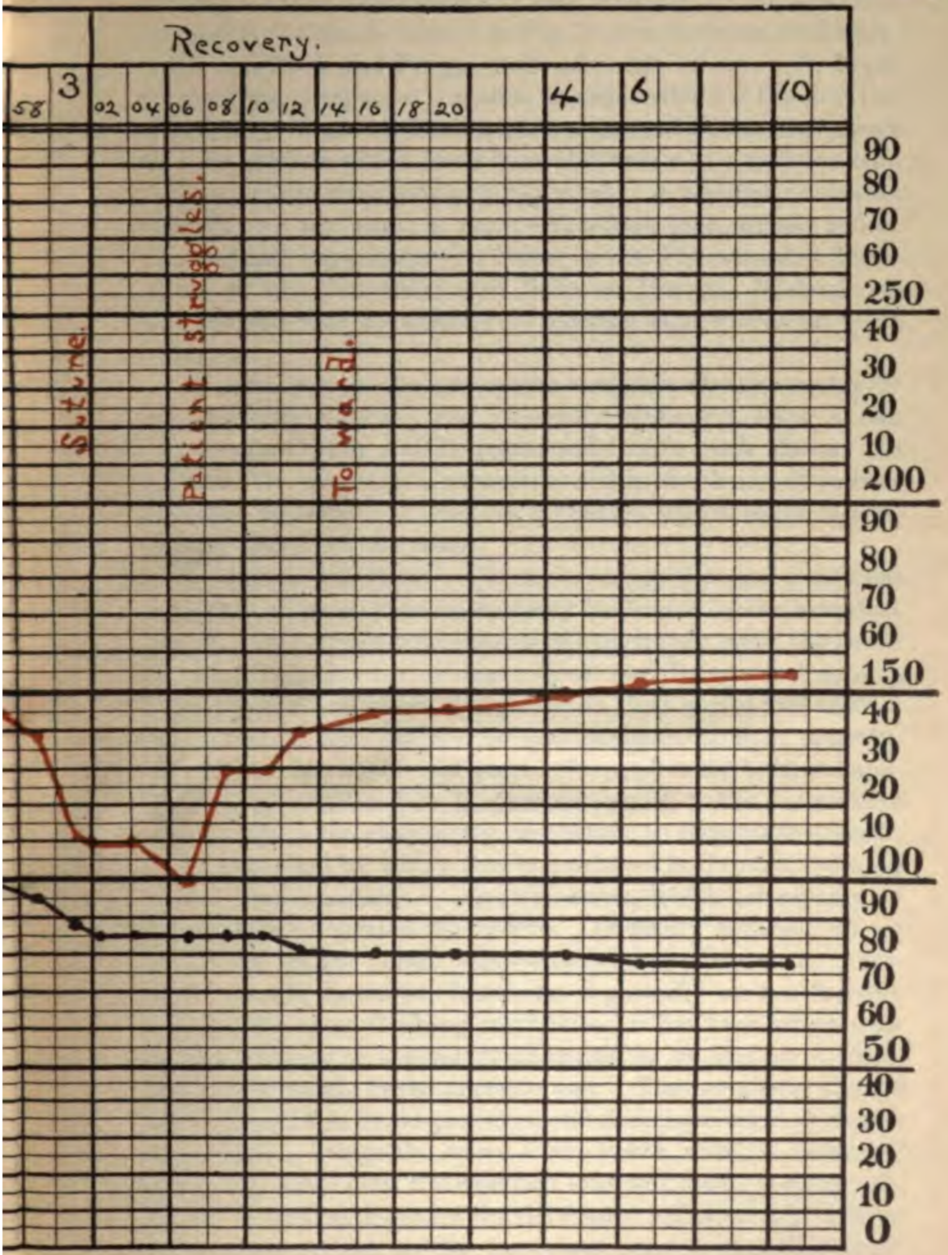


Fig. 72. (Auth

d Pulse Chart.

Prostatectomy by Dr. Eugene Fuller.

/3. Age. 60



sphygm. 12 cm.)

accomplish this, experimentally and clinically, by mechanically increasing the blood-volume, as Fig. 70 demonstrates strikingly. Crile, and Cook and Briggs, both advocate its use. In hypodermoclysis, peripheral irritation is responsible for the first rise in pressure. Dawson has supplied apparently definite evidence that, in animals which have been bled severely, a solution containing sodium bicarbonate, 0.25 per cent., in addition to sodium chloride, 0.8 per cent., is more efficacious than simple saline. Unpublished experiments by Stiles, in the Physiological Laboratory of the University and Bellevue Hospital Medical College, proved that the addition of calcium salts was of no value whatever.

In hæmorrhage, the true cardio-vascular stimulants, strychnin, digitalin, ergotin, and especially adrenalin, have a real influence, as Cook and Briggs's, and Crile's work shows. In addition, the mechanical measures used in shock are, of course, attended by benefit, as well as a posture which saves the remaining blood for the brain.

B. Collapse and Shock.—The early recognition and treatment of these conditions have been vastly facilitated by the introduction of the sphygmomanometer at the operating table, the honor of which belongs to Cushing. Cook and Briggs feel safe in stating that a fall in blood-pressure is their surest and earliest manifestation, and affords the best evidence of their severity. Crile, as we have seen (see page 157), applies the term collapse to the more sudden falls in pressure, shock to the progressive ones, in which the vaso-motor centre fails to respond to stimuli. The mechanism by which this is produced is through repeated or severe traumatisms to sensory nerves, which act reflexly on the medullary vaso-motor centre. Ordinary injuries, as we have seen, cause reflex vaso-constriction. When the vasomotor centre, however, has been repeatedly or too forcibly stimulated; or, as Cushing emphasizes, it has been affected by certain primary or secondary anæmias, as in profuse hæmorrhage, it responds in the opposite way. The peripheral stimuli call forth a fall in blood-pressure, and each successive operative manipulation brings the curve a step lower, until, in profound shock, it passes below the essential limit and death is imminent. Shock, of course, includes the depression of other activities of the nervous system, but the important point is, that the danger

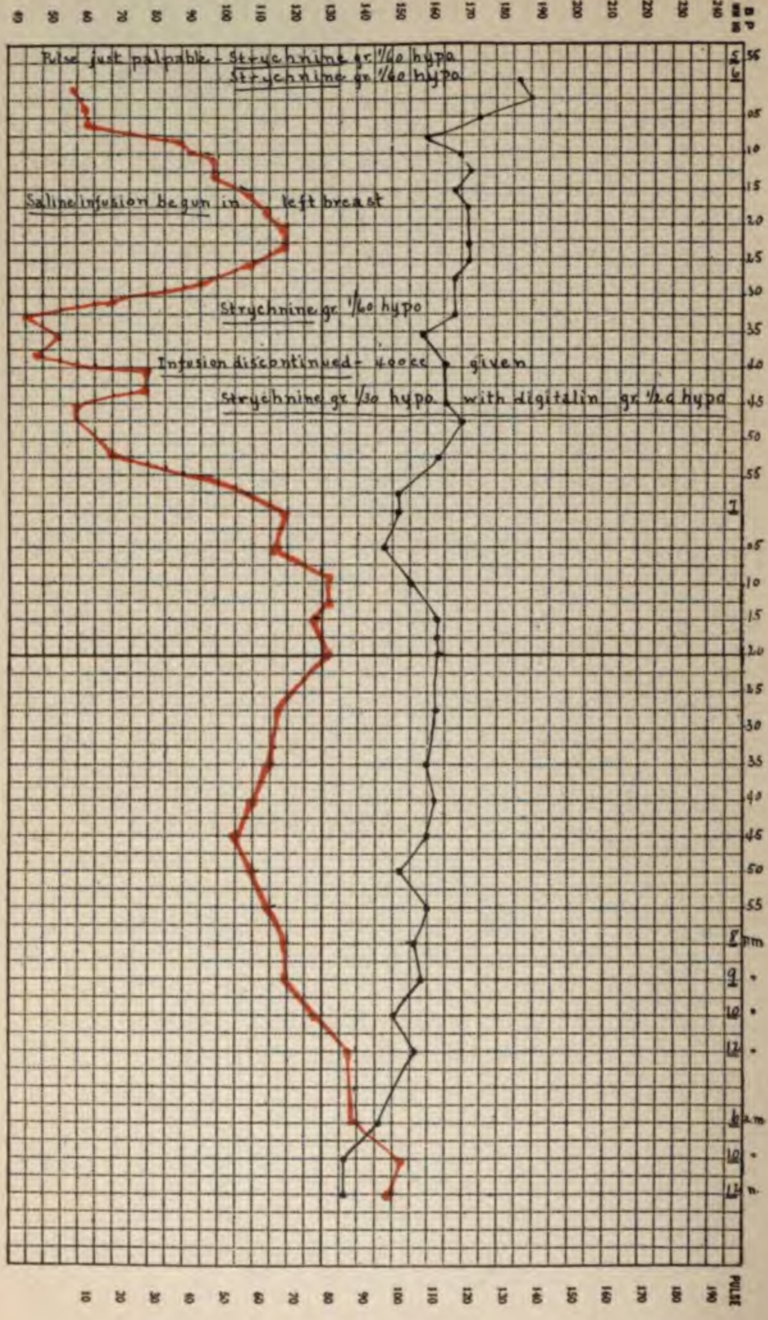

B P mm Hg
40
50
60
70
80
90
100
110
120
130
140
150
160
170
180
190
200
210
220
230
240
Pulse just palpable - Strychnine gr. 1/60 hypo
Strychnine gr. 1/60 hypo.
Saline infusion begun in left breast
Strychnine gr. 1/60 hypo
Infusion discontinued - 400cc given
Strychnine gr 1/30 hypo. with digitalin gr. 1/20 hypo
5 56
6
05
10
15
20
25
30
35
40
45
50
55
7
05
10
15
20
25
30
35
40
45
50
55
8 pm
9 "
10 "
12 "
6 am
10 "
12 n.
PULSE
10
20
30
40
50
60
70
80
90
100
110
120
130
140
150
160
170
180
190

FIG. 73.

Fig. 73.—Traumatic shock without hæmorrhage. (Cook's sphygm. 5 cm.)

Very low blood-pressures marking the depth of shock, which was otherwise clinically typical and marked.

Temporary rises in blood-pressure after each of two smaller doses of strychnin.

Marked depression of blood-pressure, with change for the worse in every symptom, during the continuance of an infusion of normal salt solution in the breast (considerable local tension).

After discontinuing the infusion, steady, progressive, and permanent rise in blood-pressure to the normal level, with complete recovery. (From Cook and Briggs, Chart No. VI.)

to life lies in the hypotension, dependent on loss of central vaso-motor, not of cardiac function.

Crile found that he could produce a state of profound shock as surely by repeated direct stimulation of the vaso-motor centre with strychnin, as he could by reflex stimulation from peripheral injury. There seems no question of the facts he has adduced.

a. **Prophylaxis.**—This includes the avoidance of all those unnecessary injuries to peripheral nerves which may call forth a depressor response; perfect hæmostasis; and the minimum manipulation of abdominal organs. Rough handling, blunt dissection, and needless expenditure of time, are dangerous, except where the blood-pressure shows favorable conditions. Chloroform should never be used where shock is feared. Cushing, and Crile, call attention to the great value of preventing the abnormal stimuli from reaching the medulla, when operations necessarily involve injury to important nerves. This is accomplished by the injection of cocain into the nerve-trunks before their division. Thus, in major amputations, the brachial plexus may apparently be divided with impunity after it has been "blocked" by cocain, and Crile always treats the superior laryngeal nerve in the same way in neck operations of any magnitude.

b. **Treatment.**—Whenever the blood-pressure shows serious depression, chloroform should be stopped and ether substituted. If ether is being used, Cook and Briggs believe its continuance safer than to allow the patient to come out, if a need for the renewal of the anæsthetic is likely to arise. All manipulations, except those essential to completing the operation at the earliest moment, should be avoided.

The question then arises as to the means of combatting the hypotension. Here the entire decision hinges upon the condition of the vaso-motor centre. Is its activity absolutely exhausted by the successive stimuli it has responded to, or is its function merely inhibited, or in abeyance? Crile seems to have demonstrated that, in profound experimental shock, the centre can no longer respond to either peripheral or central stimulation. Since he could produce shock equally by repeated injections of strychnin, he seems justified in assuming that to use such a central stimulant, when the vaso-motor centre is already exhausted by overstimulation, is positively harmful. On the other hand, it is difficult to decide clinically whether such complete exhaustion of the centre exists ; and Cook and Briggs have shown a prompt, and apparently life-saving response to strychnin and digitalin, in clinically profound traumatic and surgical shock in Cushing's wards. This has followed the use of large doses, up to gr. $\frac{1}{10}$ (see Fig. 73).

The use of saline infusion in shock is evidently worthless, and we have already seen that alcohol and ether are not hypertensive agents. How nitroglycerin and amyl nitrite, pure vaso-dilators, could be given by any intelligent surgeon, even before Crile's demonstration of their futility, is hard to understand.

Adrenalin seemed to offer a brilliant prospect of controlling blood-pressure by purely peripheral vaso-constriction. Its effects are so fugacious (see Figs. 57 and 58) that the only results so far obtained have been from using it continuously, by intravenous infusion in saline solution, 1 in 50,000 or 100,000, with addition of atropin, after Crile's method. Some sudden deaths have followed this, and Cook and Briggs have found that the general symptoms did not improve with the blood-pressure. The most rational method of maintaining the blood-pressure for long periods of time, and at any desired level, seems to be Crile's pneumatic suit. Some wonderful results have been obtained with it, and the greatest credit is due him for its invention. It deserves extensive trial, and warrants considerable hope.

c. Head Injuries.—It is impossible to enter into the complicated questions involved in the differential diagnosis of concussion, contusion, and compression of the brain, which are so admirably presented in Kocher's monograph. Measurement of the blood-pressure is of considerable service in connection with all other general and focal symptoms. A low blood-pressure may be present in concussion, as are certain other symptoms of shock; in direct injury to the medulla; and in compression, during the terminal stage. When found early after injury, it is an indication for artificial respiration and transfusion, according to Kocher, to increase the arterial blood-supply of the medulla. It is not so immediately dangerous as respiratory arrest. With hypotension, operation is not indicated for the relief of general cerebral symptoms, unless it be undertaken as a forlorn hope, in the paralytic stage of compression.

Hypertension is, as we have seen, direct evidence of anæmia of the medulla from compression (see page 141). When it supervenes after injury, it seems one of the best indications for operative interference. Cushing, in this country, has studied the matter especially, and advocates surgical interference in non-traumatic cases of general cerebral compression as well. There seems no reason why, with the blood-pressure mounting above 300 mm., an intracerebral clot should not be evacuated, as much as one from the middle meningeal artery. So far as traumatic cases are concerned, Kocher regards the appearance of the slow high tension pulse, and other symptoms of advanced compression, as evidence that the favorable period for surgical intervention has passed. The earlier diagnosis must depend on focal manifestations of local pressure, which have not yet appreciably raised the tension in the posterior fossa.

Every case of head injury, or cerebral accident, should by all means have systematic blood-pressure measurements taken at short intervals. The effects of operation are most strikingly exhibited on the blood-pressure chart (see Fig. 62).

d. Acute Peritonitis.—Crile studied the arterial tension in twenty cases of acute peritonitis from various causes, and found a moderate hypertension in all, probably dependent on reflex vaso-constriction from the peritoneal irritation. His highest reading was 208 mm., lowest 156 mm., and average 166 mm. (R. R. 5 cm.). This may be of some diagnostic value, especially in the recognition of perforative peritonitis in typhoid, which has already been considered. Late in the course, as collapse develops, the blood-pressure sinks. Heineke demonstrated this in animals (see page 156), the cause being vaso-motor paralysis similar to that in other infections.

LITERATURE

Blauel, C. Das Verhalten des Blutdrucks beim Menschen während der Aether- und der Chloroform-Narkose. Beitr. z. klin. Chirurg., 1901, vol. xxxi, p. 271.

Cook, H. W., and Briggs, J. B. Clinical Observations on Blood-pressure. Johns Hopkins Hosp. Rep., 1903, vol. xi, p. 451.

Crile, Geo. W. Blood-pressure in Surgery. Philadelphia, 1903.

—— Diagnostic Value of Blood-pressure Determinations in the Diagnosis of Typhoid Perforations. Jour. of the Am. Med. Ass'n, 1903, vol. xl, p. 1292.

Cushing, Harvey. On the Avoidance of Shock in Major Amputations by Cocainization of Large Nerve-trunks Preliminary to their Division. With Observations on Blood-pressure Changes in Surgical Cases. Ann. of Surgery, 1902, vol. xxxvi, p. 321.

—— Some Experimental and Clinical Observations concerning States of Increased Intracranial Tension. Am. Jour. of the Med. Sci., 1902, vol. cxxiv, p. 375.

—— The Blood-pressure Reaction of Acute Cerebral Compression, Illustrated by Cases of Intracranial Hæmorrhage. Am. Jour. of the Med. Sci., 1903, vol. cxxv, p. 1017.

Dawson, Percy M. Effect of Intravenous Infusion of Sodium Bicarbonate after Severe Hæmorrhage. Am. Jour. of Physiol., 1904, vol. x, Proceed. of Am. Physiol. Soc., p. xxxvi.

Duplay, S., et Hallion, L. Recherches sur la pression artérielle dans l'anæsthésie par le chloroforme et par l'éther. Arch. gén. de Méd., 1900, vol. ix, p. 130.

Frazier, C. H. A Discussion of the Surgery of Tumors of the Brain. Am. Jour. of the Med. Sci., 1904, vol. cxxvii, p. 316.

Kapsammer, Geo. Blutdruckmessungen mit dem Gärtner'schen Tonometer. Wien. klin. Wochenschr., 1899, vol. xii, p. 1279.

Kocher, Th. Hirnerschütterung, Hirndruck und chirurgische Eingriffe bei Hirnkrankheiten. Nothnagel's spec. Pathol. u. Therap., vol. ix. III Th., II Abth., Vienna, 1901.

Puricelli. La pressione sanguine in rapporto agli accidenti della narcosi chloroformica. Centralbl. f. Chirurg., 1900, vol. xxvii, p. 1318.

Remedi. Modificazioni della pressione sanguine nella operazione che si practicano nelle cavità. Centralbl. f. Chirurg., 1900, vol. xxvii, p. 93.

Schroeder, H. Untersuchungen über den Blutdruck von und nach Operationen. Münch. med. Wochenschr., 1900, vol. xlvii, p. 1437.

—— Ueber Blutdruckschwankungen nach gynäkologischen Operationen. Centralbl. f. Gynäkol., 1900, vol. xxiv, p. 1045.

CHAPTER X

BLOOD-PRESSURE IN OBSTETRICAL CONDITIONS

1. Value of the sphygmomanometer in obstetrical practice.

2. Blood-pressure during pregnancy.

3. Blood-pressure during labor and the puerperium.

- A. Labor.
  - a. Influence of chloroform.
  - b. Influence of obstetrical operations.
  - c. Influence of hæmorrhage and exhaustion.
- B. The puerperium.

4. Puerperal eclampsia.

Literature.

CHAPTER X

BLOOD-PRESSURE IN OBSTETRICAL CONDITIONS

1. VALUE OF THE SPHYGMOMANOMETER IN OBSTETRICAL PRACTICE

COMPARATIVELY little use has yet been made of the sphygmomanometer by obstetricians. During labor, the accoucheur is quite enough occupied without making blood-pressure observations. Nevertheless, one or two measurements before and after labor would be a small price to pay for the information concerning certain of the most dreaded obstetrical accidents, which can be obtained in this way. As I have said before, an intelligent nurse may easily be taught to make observations of sufficient accuracy for clinical purposes, and could keep the physician informed of the movements of blood-pressure, in cases where mishaps are feared.

The greatest importance attaches to the arterial pressure as a means of foretelling, and in consequence perhaps forestalling, an eclamptic seizure. As a measure of shock, which may accompany obstetrical operations, and of the dangerous results of hæmorrhage, it has the same value as in surgical cases. Besides these more conspicuous uses, I believe that blood-pressure determinations should be made during pregnancy whenever the urine is examined, and for the same reason. As a guide to the seriousness of a pregnancy nephritis and its liability to terminate in eclamptic convulsions, such a record would be far more adequate than the usual urinary report.

The literature contains but scanty references to studies along these lines, and few of those are with accurate instruments. Cook and Briggs's cases, so admirably shown in their charts, should stimulate investigation in lying-in hospitals. The systematic recording of blood-pressure in large series of cases ought to demonstrate its very practical usefulness.

2. BLOOD-PRESSURE DURING PREGNANCY

Before definite statements can be made concerning the influence of pregnancy on blood-pressure, there must be accurate records on the same women, at moderate intervals, throughout its whole duration. These are impossible in hospitals, where the waiting women come late, and must be furnished from private practice. At present, only measurements on different cases at different periods exist. Hence we find Queirel and Reynaud claiming that hypertension exists throughout, increasing up to labor; Wiessner, that a rise occurs during the later months; while Vaquez and Millet, Goldwater, and Cook and Briggs never found readings above the normal limit until uterine contractions had actually begun. The latter authors made the interesting observation of a fall in pressure with the descent of the fœtal head into the pelvis, in the ninth month. This seems good evidence of some previous increase in tension above the normal for the individual, from the augmented intra-abdominal pressure (see page 15). They also noted a rise in systolic pressure accompanying the painless uterine contractions during pregnancy.

The whole question is one it would be worth while to solve. The studies of Dreysel, who made weighings of the heart by Müller's method, in autopsies of seventy-six pregnant women, showed a distinct hypertrophy of both ventricles, which was proportional to the increase in body-weight. The existence of such an absolute, but not relative hypertrophy, would be no reason for assuming that hypertension would coexist, as we have already seen in discussing the hypertension of chronic nephritis (see page 146). Füth and Krönig were unable to prove increased viscosity of the blood in pregnant women. The considerable intra-abdominal tension during the last months seems to me an adequate cause for what little elevation of pressure may occur.

The most important point, at least, is settled. Real absolute hypertension, say above 180 mm. (5 cm.), 160 mm. (12 cm.), does not exist during normal pregnancy. The discovery of such tension, especially on several examinations, should be regarded as distinct evidence of mischief, and the kidneys carefully watched.

3. BLOOD-PRESSURE DURING LABOR AND THE PUERPERIUM

A. Labor.—Several factors coincide to raise the arterial pressure during labor. The excitement, the muscular effort, the contraction of the abdominal walls, the painful sensory impulses, all assist. Nevertheless, Cook and Briggs have shown that there is a constant rise in pressure during each pain, exactly parallel with the uterine contraction, and that this elevation is almost as great under full chloroform narcosis. A chart during labor shows these regularly recurring peaks on the pressure curve, which are much more striking than the slighter acceleration of the pulse. Whether due to the emptying of the uterine sinuses, and consequent increased blood-supply to the heart, or to reflex vaso-constriction, or both, does not appear. Between the pains, tension remains at a higher level than before the onset of labor. As the pains become more frequent and severe, both the average level and the maximum elevation go higher, until the child is expelled, when there is an immediate fall to somewhat below normal for the individual. Wiessner has seen a drop of 100 mm. (R. R. 5 cm.) after the birth. Vaquez and Millet, and Queirel and Reynaud give a similar picture, which seems well substantiated. Only Lebedoff and Porochjakow failed to find pressure so high during labor (v. Basch sphygm.). One great source of error exists for observations under these conditions. The voluntary contractions are not localized to the abdomen, and even the arms may participate. Even a slight heightening of muscular tone in the arm would give a fictitious value for the blood-pressure and must be avoided. Cook and Briggs's findings under chloroform, however, make it evident that the hypertension is real.

a. Influence of Chloroform.—The fact is well known, that accidents during the administration of chloroform are most exceptional in obstetrical practice, as compared with surgical anæsthesia. Cook and Briggs suggest that this may be due to the fact that hypertension during labor is the rule. Not only labor itself, but operative interference to hasten it, have marked pressor effects, which would certainly tend to counteract the effect of the chloroform. Some fall in pressure attends its use here, as in surgery, but never to a dangerous level.

Queirel and Reynaud report the lowest figures during labor as occurring during its administration.

b. Influence of Obstetrical Operations.—Instrumental delivery, version, and other methods of forced delivery seem to cause a sharp elevation of blood-pressure. According to Cook and Briggs, this "pelvic reflex" rise occurs on the introduction of the whole hand into the vagina, but is most extreme when strong traction is exerted on the child. Such hypertension makes intelligible the occasional rupture of a cerebral vessel which has occurred under these circumstances.

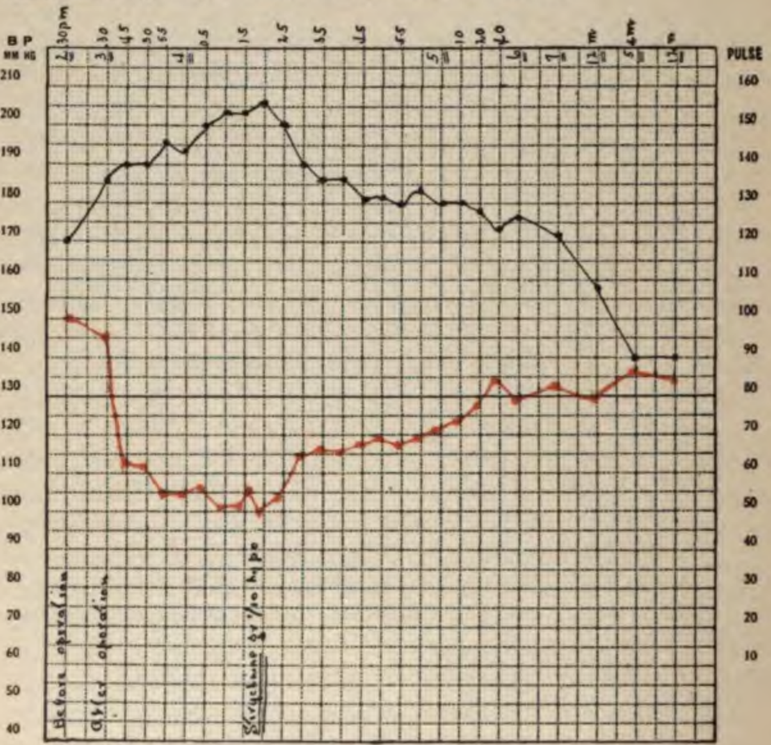


FIG. 74.—POST-OPERATIVE DEPRESSION. (Cook's sphygm. 5 cm.)

Combined shock and hæmorrhage in twin labor, after prolonged forcible attempts at instrumental delivery.

Strychnin in large amounts producing immediate and permanent rise in blood-pressure. (From Cook and Briggs, Chart No. VII.)

c. Influence of Hæmorrhage and Exhaustion.—Both loss of blood and unusual exhaustion, which may even constitute real collapse, produce hypotension. The extent of the fall in pressure, and the level at which it is maintained, are proportional to the danger that exists. Fig. 74, from Cook and Briggs, shows such a condition of hypotension and its prompt disappearance after a large hypodermic of strychnin. The blood-pressure curve here, as in other conditions of vaso-motor depression, is probably one of the best indications of the necessity for stimulation, and of its effect.

B. The Puerperium.—The changes in blood-pressure after the birth of the child must be of small consequence, and dependent on more or less individual causes. Thus Wiessner found readings lower than in pregnancy (R. R.); Goldwater, a very slight diminution in tension during the first three days, 7 mm. (G.) at the greatest; Lebedoff and Porochjakow, a fall of at least 18 mm. (v. B.) during the early days, with a return to normal during the second week; while Queirel and Reynaud report a rise in pressure, attaining its maximum as a rule on the fourth day, with subsequent slow decline.

These latter observers noted a rapid development of hypertension with any complicating inflammatory disease; peritonitis, pneumonia, or grippe. The Verdin sphygmomanometer, which they used, casts suspicion on all their results.

4. PUERPERAL ECLAMPSIA

There is no difference of opinion as to the value of the sphygmomanometer in the detection and treatment of eclampsia. Hypertension has been invariable in all the cases so far observed, as in acute uræmia. The existence of high pressure during pregnancy is a warning of its possible occurrence, which seems of more value than the finding of albuminuria. I am unable to speak from personal experience, but the testimony is so convincing and harmonizes so perfectly with what we know of blood-pressure before uræmic convulsions, that I feel assured of the wisdom of sphygmomanometric measurements as a routine during pregnancy.

During labor itself the hypertension is harder of recognition, because in large degree a normal feature. After delivery, however, if the pressure does not fall, eclampsia must be considered immanent. In all the cases studied by Cook and Briggs, this post-partum hypertension was invariably followed by convulsions sooner or later. Wiessner, Füth and Krönig, and Vaquez and Nobécourt, all insist on the great importance of this premonitory rise.

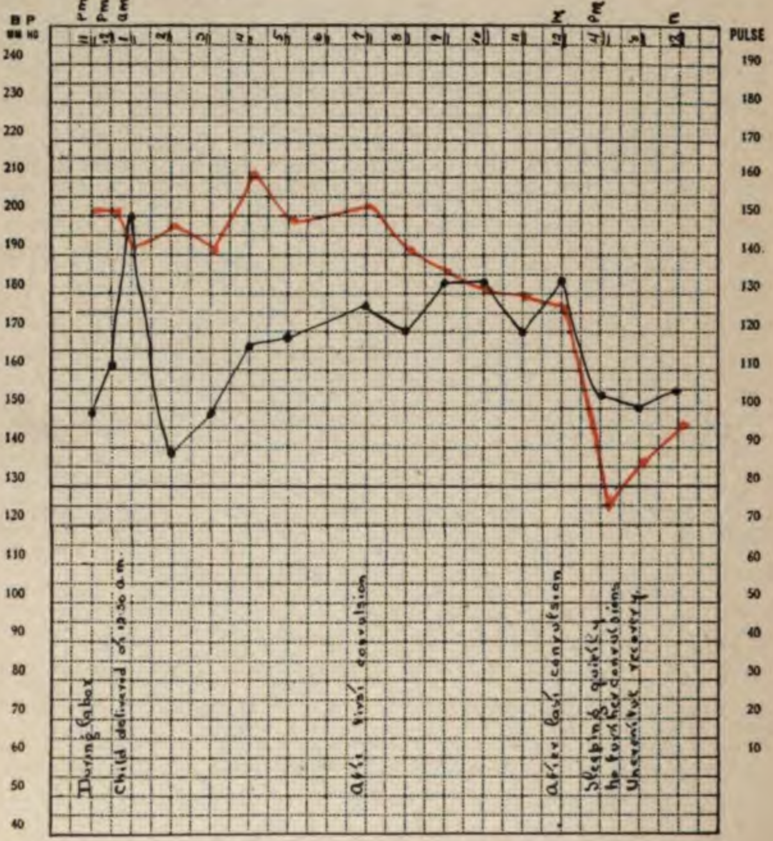


FIG. 75.—PUERPERAL ECLAMPSIA. (Cook's sphygm. 5 cm.)

Hypertension during labor.
No relief of the abnormally high blood-pressure following delivery, only a very temporary fall occurring.
With the persistence of hypertension, development of eclamptic symptoms and convulsions seven and a half hours post partum.
With the relief of hypertension (by the usual methods) disappearance of the eclamptic features. (From Cook and Briggs, Chart No. XV.)

During the actual seizure the tension reaches very high figures, Wiessner having seen it 280 mm. (R. R. 5 cm.). With its reduction by the vaso-dilators or chloroform, the convulsions disappear. Wiessner was unable to diminish the pressure by venesection and the abstraction of 200 to 300 c.c. of blood. Pal endeavors to bring eclamptic amaurosis into line with the similar transient blindness in uræmia and lead poisoning, as due to circulatory disturbance in the occipital lobe, dependent on the hypertension. He quotes no personal observation.

It is sincerely to be hoped that obstetricians in general will recognize the help which the sphygmomanometer can give them in this condition. To take the arterial tension is far easier than examining the urine, and the information thus obtained is no whit less valuable. A few measurements by the nurse, during and after labor, would give a most welcome confidence that danger from this particular quarter was unlikely.

LITERATURE

Cook, H. W., and Briggs, J. B. Clinical Observations on Blood-pressure. Johns Hopkins Hosp. Rep., 1903, vol. xi, p. 451.

Dreysel, Max. Ueber Herzhypertrophie bei Schwangeren und Wöchnerinnen. Inaug. Dissert., München, 1891. Quoted by Dienst. Arch. f. Gynäkol., 1902, vol. lxv, p. 367.

Füth und Krönig. Experimentelle Untersuchungen zur Ätiologie der Eklampsie. Centralbl. f. Gynäkol., 1901, vol. xxv, p. 701.

Goldwater, S. S. Notes on Blood-pressure in Man. Med. News, 1903, vol. lxxxii, p. 926.

Lebedoff, A., und Porochjakow. Basch's Sphygmomanometer und der Blutdruck während der Geburt und des Wochenbettes in Zusammenhange mit Puls, Temperatur und Respiration. Centralbl. f. Gynäkol., 1884, vol. viii, p. 1.

Pal, J. Zur Pathogenese der akuten transitorischen Amaurose bei Bleikolik, Urämie und Eklampsie. Centralbl. f. in. Med., 1903, vol. xxiv, p. 417.

Queirel et Reynaud. Tension artérielle et puerpéralité—grossesse—accouchement—suites de couches—physiologiques et pathologiques. XIII Congrès internat. de méd., Paris, 1900, vol. xv, Sect. d'obstetrique, p. 170.

Vaquet, H., et Millet, M. Du cœur dans la grossesse normale. La Presse Méd., 1898, vol. vi^1, p. 61.

Vaquez et Nobécourt. De la pression artérielle dans l'eclampsie puerperale. Bull. et Mem. Soc. méd. des Hôp. de Paris, 1897, vol. xiv, p. 117.

Wiessner. Ueber Blutdruckuntersuchungen während der Menstruation und Schwangerschaft. Centralbl. f. Gynäkol., 1899, vol. xxiii, p. 1335.

INDEX

Abbreviations, list of, xv.
Abdomen, operations on, 269.
  paracentesis of, 240, 269.
Abscess, of the brain, 253.
Addison's disease, 237.
Adrenalin, 223.
  in hæmorrhage, 273.
  in shock, 277.
Age, influence of, 108.
Aged, the, muscular exertion in, 123.
Albuminuria, cyclical, 183.
Alcohol, in normal man, 118.
  in disease, 223.
Amaurosis, in eclampsia, 289.
  in lead colic, 239.
  in uræmia, 182.
Amyl nitrite, in cardio-vascular disease, 211.
  in shock, 277.
Anæmia, 236.
  cerebral, 139.
Anæsthesia, general, 262.
  general, in obstetrical cases, 285.
  local, 263.
  spinal, 265.
Aneurism, 189.
  relation of, to hypertension, 152.
Anger, 119.
Angina pectoris, 202.
  treatment of, 214.
Angio-sclerosis, 148, 188.
Animals, different, blood-pressure in, 28.
Aorta, ligature of, 23.
  disease of, 143.
Aortic insufficiency, 194.
  stenosis, 196.
Apparatus, Dehio's, 208.
  Walden's, 71.
Appendectomy, 269.
Apoplexy, cerebral, 152, 189, 248.
  cerebral, operation for, 278.
Armlet, adjustment of, 56.
  influence of inner wall of, 55.
  influence of site of application of, 59.
  width of, relation to limb, 57.
  cross-section of, Hill and Barnard's, 54.
  cross-section of, Riva-Rocci's, 54.
  v. Recklinghausen's, 57.
Arrhythmia, 205.
Arteries, aneurism of, 189.
  compression of, 189.
  different, blood-pressure in, 30.
  digital, vaso-motor changes in, 68.
  dilatation of, 150.
  disease of, 143, 184.
  disease of, cerebral, 189, 248.
  disease of, treatment of, 210.
  effect of hypertension on, 150.
  hypertrophy of, 151.
  not all contracted or dilated at the same time, 24.
  rupture of, 151.
  small, dilatation of, 19.
  thrombosis of, 189.
Artery, manner of closure of, 60.
  pulmonary, blood-pressure in, 31.
  wall of, influence of, in measurements, 60.
Arterio-capillary fibrosis, 144.
Arterio-sclerosis, 143, 184.
  influence of, in measurements, 60.
Ascites, 240.
Asphyxia, 37.

Aspiration, of effusions, 240.
Asthma, bronchial, 240.
  cardiac, 204.
Attention, mental, 119.

Baths, Brand, 221.
  in normal man, 124.
  Nauheim, 210.
Basedow's disease, 200.
Blood-pressure, as a guide to adequacy of circulation, 161.
  as a guide to functional ability of heart, 161.
  diastolic, criterion of, 69.
  elements of, 136.
  end, little greater than lateral, 65.
  factors which determine, 11,
  first demonstrated, 3.
  high. (See Hypertension.)
  human, direct measurements of, 29.
  limits of, normal, 108.
  limits of, pathological, 135.
  low. (See Hypotension.)
  mean, how calculated, 4, 35, 112.
  normal, data for, 110.
  records, 101.
  relation of, to temperature, 160.
  relation of, to pulse-rate, 160.
  systolic, in brachial and digital arteries, 69.
  systolic, criteria of, 62, 64, 66.
  systolic, inspiratory fall of, 208, 230.
  variations of, cardiac, 4, 5, 32, 109.
  variations of, diurnal, 113.
  variations of, in different individuals, 108–112.
  variations of, in the same individual, 112–127.
  variations of, periodic, 32, 112.
  variations of, respiratory, 4, 5, 35, 112.
  variations of, respiratory, in asthma, 240.
  variations of, respiratory, in disease, 208.
  variations of, respiratory, in diphtheria, 230.
  variations of, spontaneous non-rhythmical, 37.
Blood-pressure, systolic and diastolic, measurement of, 129.
  systolic and diastolic, not measured by manometric trace, 5.
  systolic and diastolic, in hæmorrhage, 28.
  systolic and diastolic, in normal animal, 32.
  systolic and diastolic, relation of. (See Pulse-pressure.)
Blood, volume of, 25.
Bone, operations on, 268.
Bradycardia, 205.
Brain, abscess of, 253.
  absence of vaso-motor nerves in, 17.
  anæmia of, 139.
  arterial disease of, 189, 248.
  compression of, 139.
  cortex of, stimulation of, 21.
  diseases of, 248.
  tumors of, 253.
  tumors of, operation for, 272.
Bronchitis, chronic, 240.

Cachexia, 237.
Caffein, 139, 221.
Camphor, 139, 221.
Centres, vaso-motor, 17, 18.
  vaso-motor, in cerebral anæmia, 140.
  vaso-motor, in collapse, 155.
  vaso-motor, in shock, 157.
Charts, blood-pressure and pulse, 101.
Cheyne-Stokes respiration, 205.
  in cerebral anæmia, 140, 141.
Childhood, acute disease in, 231.
Children, normal, 108.
Chloroform, 262.
  in obstetrical cases, 285.
  in puerperal eclampsia, 288.
Cholecystectomy, 269.
Circulation, adequacy of, blood-pressure as a guide to, 161.
  arterial, general features of, 9.
  splanchnic, importance of, 22.
  splanchnic, in arterio-sclerosis, 143.
  splanchnic, in hypertension, 142.
Closure, of artery, manner of, 60.
Coagulation, method of preventing, 3.

Cocain, 263.
Cold, 124.
Colic, lead, 238.
Collapse, 153.
  experimental evidence on, 155.
  in obstetrical cases, 286.
  in pneumonia, 227.
  in surgical cases, 273.
  in typhoid fever, 219.
Color, of skin, change of, as criterion, 53, 66.
Coma, uræmic, 182.
Compensation, of the heart, disturbance of, 197, 198.
Compression, cerebral, 139.
  cerebral, operation for, 271, 278.
  circular, method of, 54–62.
  of an artery, 189.
  of limb, in measurements, influence of, 66.
Contraction, muscular, in measurements, influence of, 61.
Convulsions, epileptic, 256.
  uræmic, 182.
Coronary artery, ligature of, 12.
Cortex, cerebral, stimulation of, 21.
Cranium, operations on, 271.
Crises, vascular, in tabes, 248.
  visceral, in tabes, 247.
Criteria not in clinical use, 73.
  the several, validity of, 62–73.
  of diastolic pressure, 51, 69.
  of systolic pressure, 45, 53, 62.

Data, tabulated, for normal blood-pressure, 110.
Death, sudden, during operation, 267.
Depressor fibres in sensory nerves, 21.
Depressor nerve, 14, 21.
Diabetes, 235.
Diagnosis, of angina pectoris, 203.
  of cerebral hæmorrhage, 249.
  of collapse, in acute disease in childhood, 231.
  of collapse, in obstetrical cases, 287.
  of collapse, in pneumonia, 227.
  of collapse, in surgical cases, 273.
  of collapse, in typhoid fever, 219.
Diagnosis, of combined valve-lesions, 195.
  of epileptic coma, 256.
  of head injuries, 277.
  of lead poisoning, 239.
  of nephritis, 172, 178.
  of nephritis in pregnancy, 284.
  of peritonitis, 278.
  of puerperal eclampsia, 287.
  of shock, 273.
  of stupor, in circular insanity, 256.
  of typhoid hæmorrhage, 219.
  of typhoid perforation, 220.
  of uræmia, 182.
Diarrhœa, acute, in childhood, 231.
Digitalin, in acute disease in childhood, 231.
  in hæmorrhage, 273.
  in shock, 277.
  in typhoid fever, 221.
Digitalis, 139, 210.
Dilatation of sphincter ani, 267.
Diphtheria, 230.
Disease, blood-pressure in, 133–166.
  cardio-vascular, 169–216.
  cardio-vascular, after typhoid fever, 226.
  cardio-vascular, effect of exertion in, 208.
  of the arteries, 184.
  of the heart, muscular, 191.
  of the heart, nervous, 200.
  of the heart, valvular, 194.
Diseases, acute infectious, 216.
  acute infectious, in childhood, 231.
  chronic infectious, 231.
  chronic, with impaired nutrition, 235.
  internal, 167–244.
  mental, 253–256.
  nervous, 245–258.
  nervous, functional, 256.
Dogs, blood-pressure in, 29.
  laparotomy on, 269.

Eclampsia, puerperal, 287.
Effusion, peritoneal, 240.
  pleural, 240.

Elasticity, of the arterial wall, 24.
  of the arterial wall, necessity for, 10.
  error, in measurements, 56.
Elements, functional and essential, of blood-pressure, 136.
Embolism, cerebral, 252.
Emphysema, 240.
Empyema, operation for, 264.
Energy, of the heart, 11-16.
Endocarditis, acute, 200.
Epilepsy, 256.
Ergotin, in hæmorrhage, 273.
Error, elasticity, in measurements, 56.
Erythrol tetranitrate, in insomnia, 256.
  tetranitrate, in melancholia, 255.
Ether, 262.
  not a stimulant, 223.
  nitrous, spirits of, 211.
Excitement, 119.
Exertion, mental, 119.
  muscular, in animals, 39.
  muscular, in man, 121.
  muscular, in the aged, 123.
  muscular, in cardio-vascular disease, 208.
  muscular, in tuberculosis, 232.

Factors which determine blood-pressure, 11-28.
Fever, 160.
  typhoid, 217.
Fibrosis, arterio-capillary, 144.
Fingers, warmth of, necessary for measurements, 68.
Flow of liquids through tubes, 9.
Fright, 119.
Functional nervous conditions, 256.

Ganglion, Gasserian, operation on, 271.
Genito-urinary operations, 272.
Goitre, exophthalmic, 200.
Gout, 239.
Graphic method, 3.
Graves's disease, 200.
Gravity, influence of, in measurements, 59.
Gynæcological operations, 269.

Hæmodynamometer, Oliver's, 53.
Hæmoptysis, 234.
Hæmorrhage, cerebral, 189, 248.
  cerebral, operation for, 278.
  experimental, 26.
  in medical cases, 241.
  in obstetrical cases, 286.
  in surgical cases, 272.
  in typhoid fever, 219.
  intracranial, operation for, 271, 278.
  middle meningeal, 271, 278.
  pulmonary. (See Hæmoptysis.)
Head, injuries to, 277.
Heart, disease of, muscular, 191.
  disease of, nervous, 200.
  disease of, valvular, 194.
  disease of, effect of exertion in, 208.
  disease of, in secondary syphilis, 234.
  disease of, treatment of, 210.
  effect of hypertension on, 148.
  energy of, 11-16.
  functional ability of, blood-pressure as a guide to, 161.
  hypertrophy of, in arterio-sclerosis, 143.
  hypertrophy of, in hypertension, 148.
  hypertrophy of, in nephritis, 144.
  hypertrophy of, in pregnancy, 284.
  rate of, 13.
  reserve force of, 15.
  work of, blood-pressure as a guide to, 161.
Heat, 124.
Hemiplegia, 189, 252.
Hydrocele, excision of, 265.
Hypertension, absence of, in angina pectoris, 203.
  absence of, in arterial disease, 143, 185.
  absence of, in cardiac disease, 192.
  absence of, in nephritis, acute, 178.
  absence of, in nephritis, chronic interstitial, 172.
  absence of, in nephritis, other forms, 177.
  dangers of, for the arteries, 150.
  dangers of, for the heart, 148.

Hypertension, diagnosis of, 137.
  effect of, on the arteries, 150.
  effect of, on the heart, 148.
  essential, 142.
  essential, causes of, 142.
  false, in aortic insufficiency, 194.
  functional, causes of, 138.
  from cocain, 264.
  from ether, early stages, 262.
  in angina pectoris, 203.
  in angio-sclerosis, 148.
  in apoplexy, 189, 248.
  in arterio-sclerosis, 143, 185.
  in asphyxia, 37.
  in asthma, 240.
  in bronchitis, chronic, 240.
  in cerebral compression, 139, 278.
  in cerebral hæmorrhage, 189, 248.
  in cerebral thrombosis, 252.
  in circular insanity, 256.
  in crises, visceral, of tabes, 247.
  in diabetes, 235.
  in disease of heart, 192.
  in emphysema, 240.
  in exophthalmic goitre, 200.
  in gout, 239.
  in insomnia, 256.
  in labor, 285.
  in lead poisoning, 238.
  in melancholia, 254.
  in nephritis, 144, 169, 177, 179.
  in neurotics, 257.
  in obstetrical operations, 286.
  in peritonitis, acute, 278.
  in pneumonia, 226.
  in pregnancy, 284.
  in puerperal eclampsia, 287.
  in trigeminal neuralgia, 256.
  in typhoid perforation, 220.
  treatment of, 210.
Hypertrophy, of the heart, in arterio-sclerosis, 143.
  of the heart, in hypertension, 143.
  of the heart, in nephritis, 144.
  of the heart, in pregnancy, 284.
Hypnotics, 256.
Hypotension, causes of, 152.
  dangers of, 18, 159.
Hypotension, effects of, 18, 159.
  from chloroform, 262.
  in acute disease in childhood, 231.
  in anæmia, 236.
  in cachexia, 237.
  in cerebral compression, terminal, 141, 249, 277.
  in cerebral hæmorrhage, terminal, 249.
  in collapse, 153.
  in collapse, in obstetrical cases, 286.
  in collapse, in surgical cases, 273.
  in collapse, in typhoid fever, 219.
  in diabetes, 236.
  in diphtheria, 230.
  in general paresis, 253.
  in hæmorrhage, in obstetrical cases, 286.
  in hæmorrhage, in surgical cases, 272.
  in hæmorrhage, in typhoid fever, 219.
  in lightning pain, of tabes, 247.
  in mania, acute, 255.
  in myasthenia gravis, 253.
  in peritonitis, terminal, 278.
  in pneumonia, 226.
  in secondary syphilis, 234.
  in shock, 153, 273.
  in tuberculosis, 231.
  in typhoid fever, 217.
  terminal, 153.
  treatment of, in medical cases, 221, 231.
  treatment of, in surgical cases, 276.
Hysteria, 257.

Iodides. (See Potassium Iodide.)
Incision, 265.
Individual, normal, variations of blood-pressure in, 112–127.
Individuals, different, variations of blood-pressure in, 108–112.
Infections, acute, 216.
Influenza, 230.
Infusion, saline, in acute disease in childhood, 231.
  saline, in hæmorrhage, 272.
  saline, in shock, 277.

Innervation, double, of arteries, 23.
Insanity, 254.
  circular, 255.
Insomnia, 256.
Intestine, manipulation of, 269.
  mucous membrane of, irritation of, 269.
  resection of, 269.
Iodipin, in lead poisoning, 238.
Irregularity, of the heart, 205.

Kidneys, disease of, 169.
  disease of, amyloid, 177.
  disease of, cardiac hypertrophy in, 144.
  disease of, hypertension in, 144.
  disease of, treatment of, 210.
Kymographion, 3.

Labor, 285.
Laparotomy, 269.
Lead-poisoning, 238.
Limits, normal, of blood-pressure, 108.
  pathological, of blood-pressure, 135.
Liquids, flow of, through tubes, 9.
Locomotor ataxia, 247.

Malaria, 230.
Man, normal, blood-pressure in, 105–132.
Mania, acute, 255.
Manipulation, of nerves, in surgical operations, 267.
Manometer, mercurial, first introduced, 3.
  mercurial, errors of, 5.
  mercurial, proper calibre of, 5.
  mercurial, compensated, 6.
  elastic, 6.
  maximum and minimum, 6.
  Gumprecht's, 80.
  how filled, 103.
Marasmus, 231.
Meals, 117.
Measurement, direct, of blood-pressure, 1–6.
  indirect, of blood-pressure, 41–74.
Mediastinum, tumor of, 191.
Melancholia, 254.
Meningitis, 253.
Menstruation, in normal women, 127.
  in hysterical women, 257.
Mental activity, 119.
Method, graphic, 3.
  of circular compression, 54–62.
  v. Recklinghausen's, 73.
Methods, sphygmographic, 45, 73.
  which apply pressure through solid block, 45.
Mitral disease, 198.
Muscles, contraction of, influence of, in measurements, 61.
Myasthenia gravis, 253.
Myocarditis, 191.

Narcosis, surgical, 262.
Nephritis, acute, 178.
  chronic diffuse, 177.
  chronic interstitial, 169.
  chronic parenchymatous, 177.
Nerve, anterior crural, stimulation of, 21.
  chorda tympani, stimulation of, 19.
  depressor, 14, 21.
  sciatic, stretching, 266.
  sciatic, stimulation of, 21.
  superior laryngeal, cocainization of, 276.
  superior laryngeal, manipulation of, 267.
  vagus, 12, 20.
Nerves, manipulation of, 267.
  sensory, stimulation of, 21.
  sensory, depressor fibres in, 21.
  splanchnic, importance of, 22.
  splanchnic, section of, 23.
  vaso-constrictor, 17.
  vaso-dilator, 17, 18.
Nerve-trunks, cocainization of, 276.
  irritation of, 266.
  section of, 268.
Neuralgia, trigeminal, 256.
Neurasthenia, 257.
Neuropathic individuals, 257.
Neuroses, cardiac, 200.

Nitroglycerin, in cardio-vascular disease, 211.
  in melancholia, 255.
  in shock, 277.
Nitrous oxide, 263.
Nutrition, impaired, chronic diseases with, 235.

Obstetrics, blood-pressure in, 281–289.
Occupation, 109.
Odors, 119.
Œdema, influence of, in measurements, 61.
  of the lungs, 205.
Operations, genito-urinary, 272.
  gynæcological, 269.
  obstetrical, 286.
  peripheral, 265.
  surgical, 261.
Osteomyelitis, operation for, 267.
Output, of the ventricles, 12.

Pain, 120, 139, 256, 264.
Pains, labor, 285.
  lightning, of tabes, 247.
Palpation, of the pulse, 44.
Paracentesis, of the abdomen, 240, 269.
  of the thorax, 240, 271.
Paraldehyde, in insomnia, 256.
Paralysis, general, of the insane, 253.
  vaso-motor, 15, 18.
  vaso-motor, in collapse, 156.
  vaso-motor, in shock, 158.
Paresis, general, 253.
Pelotte, v. Basch's, 46.
  Potain's, 48.
Perforation, in typhoid fever, 220.
Peritonitis, acute, 278.
  perforative, in typhoid fever, 220.
Pertussis, 231.
Pneumatic ring, Gärtner's, 55, 82.
  suit, Crile's, in acute disease, 226.
  suit, Crile's, in shock, 277.
Pneumonia, 226.
  in childhood, 231.
Potassium iodide, in cardio-vascular disease, 211.
  in lead poisoning, 239.
Poisoning, lead, 238.
Posture, 115.
  in cyclical albuminuria, 184.
Practice, in muscular exertion, 122.
Pregnancy, 284.
Pressor effect, 21.
  fibres, 21.
Pressure, atmospheric, 38, 126.
  end, how measured, 10.
  intra-abdominal, 15.
  lateral, how measured, 10.
  total. (See END-PRESSURE.)
Prophylaxis, of shock, 276.
Prostatectomy, 272.
Prognosis, in acute disease in childhood, 231.
  in angina pectoris, 208.
  in cerebral hæmorrhage, 249.
  in diphtheria, 230.
  in general paresis, 253.
  in head injuries, 278.
  in nephritis, 173, 178.
  in pneumonia, 227.
  in puerperal eclampsia, 287.
  in surgical operations, 262.
  in typhoid fever, 217.
  in tuberculosis, 234.
  in uræmia, 182.
Pulse, palpation of, 44.
  obliteration of, as criterion, 45, 62.
  return of, as criterion, 62.
  alternating, 205
  different in two arms, 190.
  paradoxical, 208.
Pulse-rate, relation of, to blood-pressure, 13, 160.
Pulse-volume, during excitement, 119.
  during exertion, 122.
  in aged, 129.
  in disease. (See PULSE-PRESSURE.)
Pulse-wave, return of, under compression, 62.
  return of, "letting the cuff feel," 64.
Pulse-waves, rudimentary, 63.
  well-developed, 63.
Pulsation, maximum, as criterion, 51, 69.

Pulse-pressure, importance of, 35, 129.
  not measured by manometric trace, 5.
  in normal animal, 32.
  during excitement, 119.
  during exertion, 122.
  in alternating pulse, 205.
  in aortic insufficiency, 195.
  in arterial disease, 185.
  in bradycardia, 205.
  in cyclical albuminuria, 184.
  in hæmorrhage, 28.
  in hypertension, 25, 136.
  variation of, with posture, 116.
Puerperium, the, 287.
Pulmonary, artery, blood-pressure in, 31.
Psychical state, 119.
  in melancholia, 254.
Psychoses, the, 254.

Rate, of heart, 13, 160.
Records, blood-pressure and pulse, 101.
Reflex, pelvic, 286.
Reflexes, vascular, 21.
Resistance, peripheral, 10, 16.
  peripheral, blood-pressure as a guide to, 161.
Respiration, Cheyne-Stokes, 205.
  Cheyne-Stokes, in cerebral anæmia, 140, 141.
Rest, unaccustomed, 116.
Retraction, forced, 267.
Rheumatism, 230.
Rickets, 231.
Ring, pneumatic, Gärtner's, 55, 82.
  pneumatic, adjustment of, 56.
  pneumatic, width of, relation to finger, 59.

Sepsis, 230.
Sex, 108.
Shock, 153, 157, 273.
Sight perceptions, 119.
Size, bodily, 112.
Skull, fracture of, 277.
  operations on, 271.
Sleep, 116.
Sodium bicarbonate, in saline infusion, 273.
  carbonate, solution of, to retard coagulation, 3.
  nitrite, in cardio-vascular disease, 211.
Sphincter ani, dilatation of, 267.
Spinal cord, destruction of, 18.
  section of, 17.
  diseases of, 247.
Spinal canal, operations on, 271.
Spiritus etheris nitrosi, in cardio-vascular disease, 211.
Species, animal, blood-pressure in different, 28.
Splanchnic circulation, importance of, 22.
  circulation, in hypertension, 142.
  nerves, 22, 23.
  vessels, dilatation of, from depressor reflex, 21.
  vessels, constriction of, from depressor reflex, 22.
  vessels, in arterio-sclerosis, 143.
Sphygmomanometer, care of, 103.
  choice of, 102.
  development of, 46.
  leaks in, 104.
  v. Basch's, 46.
  Behier's, 45.
  Bloch's, 45.
  Cheron's, 45.
  Cook's, 80.
  Erlanger's, 93.
  Förster's, 45.
  v. Frey's, 45.
  Gärtner's, 82.
  Hill and Barnard's, 84.
  Hill and Barnard's, pocket, 53.
  Hoorweg's, 45.
  Hürthle's, 51, 73.
  Janeway's, 89.
  Landois's, 45.
  Levaschoff's, 45.
  Marey's, first, 50.
  Marey's, second, 51.
  Martin's, 80.
  Mosse's, 51.

Sphygmomanometer, Oliver's, 53.
  Philadelphien's, 45.
  Potain's, 48.
  v. Recklinghausen's, 78.
  Riva-Rocci's, 78.
  Stanton's, 87.
  Verdin's, 45.
  Vierordt's, 43.
  Waldenburg's, 45.
Sphygmomanometers, the modern, 75–104.
  comparison of results obtained with, 100.
Sphygmometer. (See SPHYGMOMANOMETER.)
Sphygmométrographe, Philadelphien's, 45.
Strychnin, in acute disease in childhood, 231.
  in collapse, in obstetrical cases, 287.
  in collapse, in surgical cases, 276.
  in collapse, in typhoid fever, 221.
  in hæmorrhage, 273.
  in shock, 276.
Suggestions, practical, for use of sphygmomanometer, 102.
Suit, pneumatic, Crile's, in acute disease, 226.
  pneumatic, Crile's, in shock, 277.
Sulphonal, in insomnia, 256.
Suprarenal extract, 228.
Superior laryngeal nerve, injury to, 267.
Surgery, blood-pressure in, 259–279.
Sweating, during anæsthesia, 262.
  during exertion, 123.
  to reduce hypertension, 214.
Syphilis, secondary, 234.

Tabes dorsalis, 247.
Tachycardia, paroxysmal, 201.
Taste perceptions, 119.
Temperament, 112.
Temperature, external, 124.
  bodily, relation of, to blood-pressure, 160.
Therapeutics. (See TREATMENT.)
Throb, subjective, as criterion, 67.
Thoracotomy, 264.
Thorax, operations on, 271.
  paracentesis of, 240, 271.
Thrombosis, arterial, 189.
  cerebral, 252.
Tissues, influence of, in measurements, 56.
Tobacco, 118.
Tone, vaso-motor, 16–24.
  vaso-motor, in collapse, 155.
  vaso-motor, in shock, 157.
  vaso-motor, variations of, local, 19.
  vaso-motor, variations of, reflex, 21.
Tonometer, Gärtner's, 82.
Traction, in operations, 267.
  on child during labor, 286.
Trional, 256, 257.
Trace, manometric, the normal, 4.
Training, physical, 122.
  physical, in cyclical albuminuria, 184.
Transfusion, 26, 28.
Traube-Hering waves, 36.
  in man, 112.
  in cerebral anæmia, 140, 141.
Treatment, of acute disease, 221.
  of acute disease in childhood, 231.
  of cardio-vascular disease, 210.
  of hæmorrhage, 272.
  of hypertension, 210.
  of shock, 276.
  of typhoid fever, 221.
Tuberculosis, 231.
  in childhood, 231.
Tubes, flow of liquids through, 9.
Tumor, of the brain, 253.
  of the brain, operation for, 272.
  of the breast, operation for, 268.
  of the mediastinum, 191.
Typhoid fever, 217.
  late effects of, 226.

Uræmia, 182.
  treatment of, 214.
Uterus, operations on, 269.

Vagus, stimulation of central cut end of, 20.

Vagus, stimulation of peripheral cut end of, 12.
Variations, of blood-pressure. (See BLOOD-PRESSURE.)
Vaso-constrictor nerves, 17.
Vaso-dilator nerves, 17, 18.
  drugs, in cardio-vascular disease, 211.
  drugs, in puerperal eclampsia, 288.
  drugs, in shock, 277.
Vaso-motor centres, 17, 18.
  centres, in asphyxia, 37.
  centres, in cerebral anæmia, 140.
  centres, in collapse, 155.
  centres, in shock, 155, 157.
  paralysis, effect of, on heart, 15.
  tone, the, 16–24.
  tone, in collapse, 155.
  tone, in shock, 157.
  tone, variations of, local, 19.
  tone, variations of, reflex, 21.
Velocity of stream, a component of end pressure, 10.
Venesection, in cardio-vascular disease, 214.
  in puerperal eclampsia, 289.
Ventricle, right, maximum pressure in, 32.
Ventricles, output of, 12.
Verification, of criterion of maximum pulsation, 69.
  of criterion of return of color, 66.
  of criterion of return of the pulse, 62.
  of method of circular compression, 55.
Vertebræ, operations on, 271.
Vessels, cerebral, disease of, 189.
  splanchnic, 21, 22.
  splanchnic, in arterio-sclerosis, 143.
  splanchnic, in hypertension, 142.
Volume, of the circulating blood, 25.
  output of the ventricles, 12.

Waves, Traube-Hering, 36, 112.
  Traube-Hering, in cerebral anæmia, 140, 141.
Work, muscular, 30, 121.
  muscular, in cardio-vascular disease, 208.
  muscular, in the aged, 122.

(1)

THE END

THE PRINCIPLES AND PRACTICE OF MEDICINE.

By WILLIAM OSLER, M. D.,

Fellow of the Royal College of Physicians, London; Professor of Medicine in the Johns Hopkins University, and Physician in Chief of the Johns Hopkins Hospital, Baltimore; formerly Professor of the Institutes of Medicine, McGill University, Montreal; and Professor of Clinical Medicine in the University of Pennsylvania, Philadelphia.

THE WORK HAS BEEN REVISED, REWRITTEN, RESET, ENLARGED, AND BROUGHT UP TO DATE IN ALL DEPARTMENTS.

Fifth Edition. 8vo, 1182 pages. Cloth, $5.50; half morocco, $7.00.

Sold only by Subscription.

In preparing the revision the author has depended not only upon his own inexhaustible resources, but has been assisted by men who are especially qualified in certain lines of medical practice and in pathological investigation. He has in all sections tried to maintain the thoroughly practical character of the work, as a guide in diagnosis, symptomatology, and treatment. The work is already the standard text-book of nearly every reputable medical college in this country, being recognized and accepted as a safe, sure, and scientific teacher for the student of medicine, as it is also an indispensable work of reference and a reliable guide to the physician who wishes to occupy a place in the front rank of medical practice.

"The first edition was great, the second greater, and this last greatest of all."—*Cincinnati Lancet-Clinic.*

"There are few books which receive professional confidence and esteem to the extent of that received by Dr. Osler's well-known work, for three large editions have been presented to us in a period of about six years."—*Therapeutic Gazette.*

"We have little to add to our notice of the second edition (*vide* Journal, November 30, 1895, page 966). The criticisms then made hold good now, but we regarded the work as one of the ablest contributions to the literature of the subject in this country—an opinion which we still hold."—*Journal of the American Medical Association.*

"The work, like former editions, is thoroughly practical in its character, fully up with the times and down to date, and is a safe guide to all practitioners who may consult its pages. The student of medicine, whose time is so much occupied by the tremendous amount of knowledge he is expected to master before graduation, will find here exhaustiveness without verbosity and a plain matter-of-fact method of discussing the characters of different diseases, that can not fail to instruct without wearying."—*Journal of Medicine and Science.*

D. APPLETON AND COMPANY, NEW YORK.

THE SURGICAL DISEASES OF THE GENITO-URINARY ORGANS.

By E. L. KEYES, A. M., M. D., LL. D.,

Consulting Surgeon to the Bellevue and the Skin and Cancer Hospitals; Surgeon to St. Elizabeth Hospital; formerly Professor of Genito-Urinary Surgery, Syphilology, and Dermatology at the Bellevue Hospital Medical College, etc.; and

E. L. KEYES, Jr., A. B., M. D., Ph. D.,

Lecturer on Genito-Urinary Surgery, New York Polyclinic Medical School and Hospital; Assistant Visiting Surgeon to St. Vincent's Hospital; Physician to the Venereal Clinic, Out-Patient Department of the House of Relief of the New York Hospital, etc.

Cloth, $5.00; half leather, $5.50. Sold only by Subscription.

WITH ONE HUNDRED AND SEVENTY-FOUR ILLUSTRATIONS IN THE TEXT AND ELEVEN PLATES, EIGHT OF WHICH ARE IN COLORS.

"It is certainly refreshing that a man of the experience and ability of the author of this volume has culled from this mass of literature the essentials, and given us in a well-planned volume the gist of the entire subject.

"The book is systematically arranged, and each subject is taken up and dealt with in a way that makes it easily accessible to the busy practitioner. The style is explicit and never verbose, which, with the fine vein of humor running through it, makes it very enjoyable reading."—*Northwestern Lancet.*

"Gonorrhea is gone into more extensively than in any other work."—*Denver Medical Times.*

"As a text-book on Genito-Urinary Surgery it stands at the head of the publications on the subject in the English language. Our readers wishing an up-to-date work on the subject can not do better than to buy this latest and newest work."—*Medical Century.*

"The book is well illustrated, well printed, well arranged, and will be more popular than its predecessors."—*Chicago Medical Recorder.*

"The chapters on the affections of the posterior urethra, prostate and seminal vesicles are especially good, and many 'pointers' are found in the chapter on the 'Treatment of Urethral Inflammation and their Immediate Complications.'"—*Canada Medical Record.*

"This is a good book on an important subject. Within the compass of 800 pages it gives a comprehensive treatment of the various diseases of this special branch of surgery, and, while not discarding the good of the older work, it embraces all that is new in this field."—*Journal of Medicine and Science.*

"This is so well written as to be exhaustive in character, and needs little or no comment from the reviewer further than to mention its completeness in every particular."—*Cincinnati Lancet-Clinic.*

D. APPLETON AND COMPANY, NEW YORK.

"A TECHNICAL BOOK OF BROAD USEFULNESS."
—THE TIMES, NEW YORK.

THE LAW IN ITS RELATIONS TO PHYSICIANS

By ARTHUR N. TAYLOR, LL.B.

Of the New York Bar

NEW EDITION, REVISED AND ENLARGED

12mo. Cloth. Price, $2.00

" Rarely has it been our privilege to examine a book which so clearly seems to just fill a demand as this one does. The physician must ever and again recur to the thought that by some slip he may expose himself to a suit whose loss will mean his financial ruin. The average physician does not know in any adequate way just what can legally be expected of him by his patient. Nor can he find out without great effort and probably expense. Neither, on the other hand, does he know what rights he has in the courts. If called into court he must submit utterly to the advice of his attorney. It is to meet these conditions that this book is issued. The author quotes fully of cases, decisions and opinions of leading courts and jurists everywhere, but especially in the various states. It is information such as the physician needs in his ordinary practice in order to protect himself, his reputation, and his family. Of course, unusual cases will come up, but if we know the principles underlying them we may act much more wisely. We believe the book will have a very wide circulation here in New England, a success which it has already achieved in New York."—*Annals of Gynæcology and Pediatrics, Boston.*

" Every physician should, unless conversant with the law, avail himself of the contents of this book, and he will find himself better prepared to meet the conditions and circumstances involving legal complications into which any of us are liable to find ourselves drawn."
—*Journal of Nervous and Mental Diseases, New York.*

" This volume is indeed a most valuable one, because physicians should have an idea of the law as it regards them. For often it is found that physicians are ignorant of the most elementary principles of the law; and this is not all, they do not understand the law which refers to them. This book is an excellent treatise and one which brings out all the facts which it is important that a physician should know. We advise all physicians to buy this book and read it through. In a thousand and one instances it will be found of the greatest value."—*Medical Progress, Louisville.*

D. APPLETON AND COMPANY, NEW YORK.

GYNÆCOLOGY

A Text-Book for Students
and a Guide for Practitioners

By WILLIAM R. PRYOR, M.D.

Professor of Gynæcology in the New York Polyclinic Medical School; Attending
Gynæcologist New York Polyclinic Hospital; Consulting Gynæcologist St.
Vincent's Hospital, New York City Hospital, and St. Elizabeth's
Hospital; Membre Fondateur Congress International de
Gynæcologie et d'Obstetrique; Fellow of the American
Gynæcological Society; Fellow New York
Academy of Medicine

Sold only by Subscription. Cloth, $3.50

"The author of this book has had a large experience, both as a teacher and an operative surgeon, and we would expect him to produce an excellent treatise. This he has undoubtedly done, but he has also accomplished more, for the book is especially well written, being very readable, and at the same time clear and concise, so that both students and practitioners will find it a valuable guide in this important field. The division of the work into two parts is admirable, the first treating of diseases and the second giving the operative procedures, as it enables the reader to quickly find what he is looking for. The chief features of the book are the avoidance of extraneous topics, the omission of minute anatomy and bacteriologic examinations, and the prominence given to non-operative measures as well as operative treatment. The illustrations are many and artistic, and add to the value of the text, and the general make-up of the book is a credit to this well-known firm of publishers. Those who buy this book will not be disappointed in it."—*Journal of Medicine and Science.*

"The literary style is good. The author has an easy and graceful mode of expression, and thereby greatly enhances the usefulness of his book. The illustrations are very artistic and are mostly original. It has not been our fortune to examine any book that will better repay study than this one from the pen of Dr. Pryor. We congratulate both author and publisher upon the results of their efforts to give the medical profession a really good book.

"Everything is found in it that is likely to be met with, even by the specialist. He devotes considerable space to the treatment of disease, and the performance of operations by the vaginal route, yet he admits that many cases arise where the abdominal route is alone suitable. He therefore gives both methods fully."—*Canada Lancet.*

D. APPLETON AND COMPANY, NEW YORK.

LANE LIBRARY. STANFORD UNIVERSITY

LANE MEDICAL LIBRARY

To avoid fine, this book should be returned on or before the date last stamped below.

JUL 30 1964

SEP 4 1968

~~JAN 1 8 1977~~

L74 Janeway T.C. 47663
J33 Clinical study of th
1904 blood-pressure

NAME	DA
Newton Harland SM III	
[illegible]	

L74
J33
1904